

MILITARY SURGICAL MANUALS
NATIONAL RESEARCH COUNCIL

VOLUMES IN THIS SERIES

- I MANUAL OF STANDARD PRACTICE OF PLASTIC AND MAXILLO-FACIAL SURGERY**
- II OPHTHALMOLOGY AND OTOLARYNGOLOGY**
- III. ABDOMINAL AND GENITO-URINARY INJURIES**
- IV. ORTHOPEDIC SUBJECTS**
- V. BURNS, SHOCK, WOUND HEALING, AND VASCULAR INJURIES**
- VI NEUROSURGERY AND THORACIC SURGERY**

FOREWORD

THE Medical Department of the Army has been confronted with the necessity for enormous and rapid expansion paralleling that of the armed forces. The state of war has greatly increased the task of furnishing adequate medical care for Army personnel since battle casualties are added to the already wide range of diseases and injuries that must be treated.

Expansion of the medical establishment of the Army is entirely dependent on entry into the service of individuals from civil life. While most reserve officers have had a varying amount of military training, practically all medical officers will encounter problems in the military service entirely foreign to their previous experiences. These problems are by no means confined to those of an administrative nature, many are distinctly professional. The military situation imposes certain restricting factors which render impracticable some procedures that would be considered ideal in civil life. The goal of furnishing the best possible treatment to all individuals is the same in the Army as in civil life, but the means to attain that goal may differ materially.

There has been a marked tendency to specialization within the medical profession since the first World War. This tendency is fundamentally sound but does serve to increase the problems of many individual medical officers in time of war. Specialization cannot be followed to the same degree in the military service as in civil life. While many highly qualified specialists in the various fields of medicine and surgery will serve in like capacities in the Army this cannot invariably be true. The great burden of medical care will fall on medical officers outside the highly specialized fields. It is thus essential that nearly all medical officers be familiar with the principles of military surgery. Recent advances in therapy have resulted in radical modification of certain principles of treatment that were formerly considered sound.

This series of texts presents in compact form essential up-to-date and reliable information regarding military surgery. The various sections have been written by outstanding authorities in their respective fields. They have been prepared for publication under the auspices of the Division of Medical Science of the National Research Council.

These texts will prove a highly valuable source of professional information for any surgeon desiring a knowledge of the principles of military surgery. Their application is not confined to military medicine, for most of the wounds and injuries of modern warfare may be duplicated in civil emergencies. The condensed form and avoidance of debatable points will render them very convenient for quick reference as well as for more mature study.

These volumes represent an important addition to the field of surgical texts. The individuals instrumental in their preparation have made a distinct contribution to civil and military medicine by their assemblage and presentation of this timely professional information.

JAMES C. MAGEE

Major General, U. S. Army
The Surgeon General

The naval medical officer is often faced with medical or surgical situations with which he must deal entirely alone and without the opportunity for consultation and assistance from other members of his profession. He may be the only medical man on a ship in the middle of an ocean, and any surgical emergency must be met by him and him alone. He cannot refer the case to a specialist; he himself must do everything that is necessary. It is important that he have the best assistance that professional books and journals can give him. A volume such as this, which contains practical and essential things, readily accessible, is a real help to a medical officer and patient in this situation.

ROSS T. MCINTIRE

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Surgeon General, U. S. Navy

BURNS, SHOCK WOUND HEALING AND VASCULAR INJURIES

*Prepared under the Auspices of the Committee on Surgery
of the Division of Medical Sciences of the
National Research Council*

ILLUSTRATED

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INTRODUCTION

THIS volume is one of a series developed under the auspices of the Division of Medical Sciences of the National Research Council to furnish the medical departments of the United States Army and Navy with compact presentations of necessary information in the field of military surgery. The individual manuals are prepared under the auspices of the various subcommittees of the Committee on Surgery of the Division of Medical Sciences of the National Research Council and are edited by the Committee on Information.

The first five volumes cover the following subjects: plastic and maxillofacial surgery; ophthalmology and otolaryngology; abdominal and genito-urinary injuries, orthopedic subjects; and burns, shock, vascular injuries, and wound healing. The sixth volume contains material on neurosurgery and thoracic surgery.

The Committee on Surgery includes Drs. Everts A. Graham, Chairman, Irvin Abell, Donald C. Balfour, George E. Bennett, Warren H. Cole, Frederick A. Collier, Robert H. Ivy, Herman L. Kretschmer, Charles G. Mixer, Howard C. Naffziger, Alton Ochaner, I. S. Ravdin, and Allen O. Whipple. The Committee on Information includes Drs. Morris Fishbein, Chairman, J. J. Bloomfield, John F. Fulton, Richard M. Hewitt, Ira V. Hiscock, Sanford V. Lerkey, and Robert N. Nye.

Most of the detail of the editorial work has been done by Dr. Richard M. Hewitt, head of the Division of Publications, the Mayo Clinic, Rochester, Minnesota.

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BURNS

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PREFACE

THE treatment of burns should be a matter of teamwork. A carefully trained "burn team" that is accustomed to cooperative efforts in periods of relative inaction will be better fitted to handle the increased burden of heavy casualties and to maintain the very important morale of the burned victims. Adequate function of a burn team requires the concerted efforts of those trained in general surgery, physiology, plastic surgery, and laboratory work. A surgeon with physiologic training or aptitudes makes the best leader of a burn team.

The writing of this manual also represents the efforts of a group. In its preparation I have been assisted by two members of our own burn team at the Henry Ford Hospital. To make the work more complete and authoritative I called on Dr James Barrett Brown of Washington University, St. Louis, to prepare the chapter on late plastic care. The chapter on burns in chemical warfare was prepared by Dr David P Barr of Cornell University Medical College. This manual represents, then, the efforts not only of several individuals, but of three institutions.

ROY D McCLURE

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BURNS

CHAPTER I

THE GENERAL TREATMENT OF THE PATIENT WITH A SEVERE BURN

HENRY N HARKINS M.D

THERE is, perhaps, no condition in which the general treatment of the patient is of such importance as in burns. The statement of Pack and Davis in their book, entitled "Burns," that "it is penny-wise and pound-foolish to consume invaluable time in applying perfect local

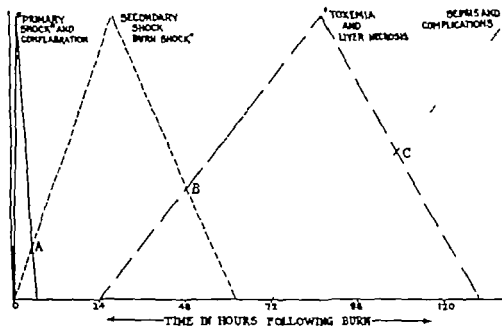


CHART SHOWING OVERLAPPING OCCURRENCE OF
MAIN CAUSES OF BURN DEATH

From 0 to A primary shock is predominant From A to B secondary shock
From B to C toxemia and after C sepsis and complications

(Modified from Green and Hollmann)

Fig. 1.—Chronology of chief causes of death from burn.

dressings while the patient is sinking into irrecoverable shock," is apropos. Local treatment must be used in such a manner that it does not interfere with, but rather complements and supplements, general treatment.

As is shown in Fig. 1, deaths from burns occur during four overlapping stages. (1) "primary shock," (2) shock, (3) toxemia, and (4) infection

The general treatment of burns should be directed against death during these four stages. Since these phases occur so rapidly, the general treatment of burns is really a continuous process and can be roughly divided under the following headings: (1) supportive measures, (2) treatment and prevention of shock, (3) prevention of toxemia, (4) prevention and treatment of infection, and (5) general care of old burns

SUPPORTIVE MEASURES

Rest and Quiet

To supply these conditions is advisable

Elevation of Feet

This is useful as a temporary, emergency measure if shock is present.

Warmth

In excess, warmth may be harmful. So-called burn-tents should not be kept hotter than 85° F

Sedatives

These are useful when given on the patient's admission, but later they may exaggerate oxygen lack. The present trend in the United States is to perform less radical débridement of burns than is the custom in Great Britain. With such minor débridement, general anesthesia is seldom necessary, hence, sedatives are of increased importance in production of analgesia and there is a consequent necessity for large doses. When an adult of normal size has sustained severe burns as much as $\frac{1}{2}$ grain (0.032 gm.) of morphine sulfate in a single dose may be administered with impunity before local treatment is begun. For use in the British Navy, Wakeley (1941) stated concerning "the early administration of morphia in adequate doses," that "men require $\frac{1}{3}$ gr. and this should be repeated if pain is not relieved." Wallace (1941), writing in his *British War Manual on Burns*, suggested the following two dosages of opiates for adults:

Tinct. Opi
Inj Morph

M. XXX
gr $\frac{1}{4}$ to $\frac{1}{2}$

Stimulants

Probably these are of value only when respiratory depression is present.

Vasospastic Substances

These are mainly symptomatic remedies.

TREATMENT AND PREVENTION OF SHOCK

This is one of the chief items of all treatment of burns. The importance of "burn shock" is shown in Chart I. Various estimates have put the percentage of deaths that are due to shock at from 60 to 75 per cent of the total number of deaths in cases of burns. Wilson

The Mortality of Severe Burns

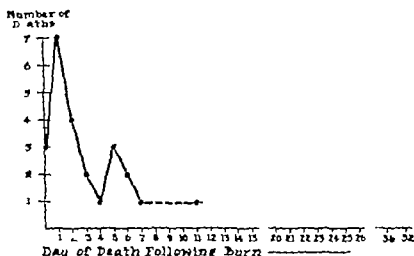


Fig. 2.—The preponderance of deaths from burns in the first two days. This graph demonstrates that in a series of thirty fatalities from burns occurring at the Pennsylvania Hospital in the past decade, fourteen occurred in the course of the first forty-eight hours (Rhoads, Wolff and Lee: *Ann. Surg.*, June, 1941. J. B. Lippincott Co.)

(1928) reported that 63 per cent of his eighty fatalities in cases of burns, occurring from 1913 to 1925, were from shock; and Seeger (1937) reported that 64 per cent of his total deaths in cases of burns were attributable to shock. The most recent figures in this regard are those of Atkins (1940) who had considerable experience in treating burned patients evacuated from Dunkerque. This writer stated "Secondary shock is the most serious factor in burns and is responsible for 60 per cent of the deaths occurring in this condition." Naturally with adequate therapy the figure will not run this high, but the fact still remains that about two-thirds of untreated burned patients will die of burn shock.

CHART 1

THE IMPORTANCE OF BURN SHOCK

- 1 Approximately 6000 persons die from burns in the United States every year
- 2 Of these deaths, about 60 to 75 per cent are due to burn shock. Atkins, who had an extensive experience with burned men evacuated from Dunkerque, said recently "Secondary shock is the most serious factor in burns and is responsible for 60 per cent of the deaths occurring in this condition."
- 3 It is thus seen that burn shock is of extreme practical importance in time of war and even in peace time is responsible for about 4,000 deaths annually in this country alone
- 4 Of all the causes of burn fatalities, shock is not only one of the easiest to cure, but also to prevent.

Pathologic Physiology of Burn Shock

While there is considerable dispute on certain points, most investigators will agree to the following outline of the essential processes in burn shock: The local thermal trauma produces a local capillary injury with regional loss of plasma-like fluid both into the burned tissues and from the skin by means of "weeping." Later, there may be generalized infiltration of plasma into tissues remote from the region of the burn. The quantitative aspect of the loss of plasma is of especial significance, the loss may exceed several liters. Resulting from the loss there occurs reduction in blood volume, cardiac output and blood flow, with associated hemoconcentration, as is shown in Chart 2. Correction of this condition before dangerous and irreparable anoxic damage to tissue and irreversible, generalized increased capillary permeability occur, forms the prime element in the treatment of burn shock.

Time of Occurrence of Burn Shock

In serious cases, much of the plasma may be lost in the first hour after a burn. The need for haste in beginning treatment is thus evident. Arbitrarily burn shock, as is shown in Figs 1 and 2, is considered to be most important during the first forty-eight hours after a burn.

Relation of Shock to Local Treatment (Chart 3)

Tannic acid and other tanning agents have done more to reduce the mortality during the first forty-eight hours than during the later course in cases of burn. Whether the tanning method does this by sealing off the damaged tissues and preventing loss of fluid or by

CHART 2

SUMMARY OF NATURE OF BURN SHOCK

I. Cause

- A. The local thermal injury produces a regional increase in capillary permeability with loss of plasma-like fluid into the tissues and to the outside by "weeping."
- B. Later an irreversible stage arrives with generalized increased capillary permeability and tissue damage. Therapy should be begun before this stage.
- C. The loss of plasma is the chief factor and is the chief indication for treatment. It produces:—
 1. Hemoconcentration.
 2. Decreased blood volume.
 3. Decreased cardiac output.
 4. Decreased blood flow.
 5. Secondary vasoconstriction.
 6. Secondary fall of blood pressure.

Hemoconcentration is the most important diagnostic and prognostic sign in uncomplicated burns.

Blood chemical changes are essentially secondary and include:—

1. Increase in plasma K Mg^{++} Ca^{++} and in blood nonprotein nitrogen and bilirubin.
2. Decrease in plasma Na Cl and protein, and in blood sugar.

II. Time of onset

The processes that lead to shock begin at the time of the burn.

III. Duration

Death during the first 48 hours after the burn is usually due to shock.

IV. Extent of loss of plasma in cases of burn

This may reach the alarming figure of several liters.

V. Relation to other causes of burn deaths

- A. Death from the 49th to the 96th hour is usually due to "toxemia and hepatic necrosis."
- B. Death after the 120th hour is usually due to sepsis or other complications.

precipitating and diminishing the absorption of toxic products is still uncertain. The bald fact remains, however, that during this period the mortality has been markedly diminished according to most reports. Even granting, then, that certain other local remedies may give a better cosmetic result on the face or hands, it would seem that in cases of severe burn, local treatment should be chosen with an eye toward the prevention of shock and saving of the life of the patient rather than only to the ultimate cosmetic result. Tanning should be applied promptly in cases of severe burn when adequate plasma is not available oils and saline dressings or baths should be reserved

CHART 3

RELATION OF SHOCK TO LOCAL AND GENERAL TREATMENT

A Relation to local treatment.

In treatment of burns saving the life of the patient is more important than the ultimate cosmetic result. In this connection, Pack and Davis (1930) stated

"It is penny-wise and pound-foolish to consume invaluable time in applying perfect local dressings, while the patient is sinking into irrecoverable shock."

Rapid tanning to prevent loss of fluid is the best local treatment from the standpoint of preventing shock in cases of serious burn.

B Relation to general treatment of other complications. Glucose prevents hepatic necrosis. Asepsis and, in certain cases, specific treatment with sulfonamides, as prophylaxis against sepsis should be considered along with the treatment of shock itself

Administration of dye, as advised by Aldrich (1933), of sulfonamides by mouth as advised by Penberthy and Weller (1939) and of sulfonamides locally as advised by Hooker and Lam (1941) is of value in this connection

in such instances for mild extensive, or localized deeper, burns. When adequate plasma is available, other factors may be more important and the danger of hepatic necrosis from absorption of tannic acid may outweigh the hazards of increased loss of plasma. This is especially true since hepatic necrosis cannot be adequately treated, whereas the plasma lost may be restored. Furthermore, the pressure-dressing method of treatment of burns as used by Koch actually prevents loss of plasma by physical rather than by chemical means.

All the time that local treatment is being given, measures for control of shock should be carefully carried out. Thus, on admission, even before tanning or other local measures have begun, a specimen of blood should be obtained for typing and cross-matching (in cases wherein whole blood is to be given) and for estimation of blood concentration by hematocrit. This enables the determinations to be started while local therapy is being carried out.

Desirable Determinations

The course following a severe burn should be ascertained by the following determinations:

1. Blood pressure should be charted every two hours for the first forty-eight hours and every eight hours for the next five days unless the systolic pressure is less than 90 mm. of mercury; then more frequent readings are necessary

2. Temperature, pulse rate and respiratory rate should be determined every two hours.

3. A chart should be kept of the daily intake and output of fluid, together with the specific gravity of each specimen of urine. Making of these records should continue through the first three days, and later if the daily output is less than 1200 cc.

4. The value for hemoglobin should be estimated or a hematocrit determination should be made every three hours during the first twelve hours and thereafter if the value is elevated. In any event, one or the other of these determinations should be made daily for the first week. (Later when secondary anemia develops, as it does almost invariably following severe burns, these same determinations are of great value in the control of this late complication.)

5. Daily leukocyte counts, urinalysis and blood chemical determinations (plasma proteins, chlorides, nonprotein nitrogen, carbon dioxide combining power, and icteric index) should be made.

Except in large hospitals, or during periods of relative inaction at the front, it is difficult to obtain all these determinations. If only one were to be made, there is no doubt as to which would be chosen the hematocrit determination (or the value for hemoglobin or erythrocyte count). No other single test affords anywhere near as much information concerning the status of a patient who is in the state of burn shock. In the presence of burns, hemoconcentration, and not fall in blood pressure, is, as a rule, the first indication of impending shock and of the consequent necessity of replacement of fluid. In practically no other type of shock does hemoconcentration serve such a useful rôle in diagnosis and prognosis and it behooves the surgeon to take advantage of this.

General Treatment

The treatment of such shock consists in the general supportive measures that have been listed (p. 4) supplemented by three forms of specific therapy—oxygen, adrenal cortical extract, and fluid replacement therapy.

Oxygen.—The importance of anoxia in shock has been recognized for some time, but the work of McClure, Hartman, and their associates (1935–1939) has reemphasized its significance. Wakeley (1941) gave the following advice relative to burns in the British Navy: "Oxygen administered by a Boothby mask enables the patient to obtain this in the alveolar air. In cases which are complicated by 'blast lung' this form of administering oxygen is most advantageous. In the Navy

where Boothby [B. L. B.] masks have been in use for all badly shocked burn cases, their value has been inestimable"

Mutch (1940) analyzed methods of administration of oxygen. He stated that the tube and funnel method did not appreciably raise the oxygen content of the alveolar air. The other three methods in common use, in which nasal catheters, tents or the B.L.B. mask are used, effect a considerable rise in the alveolar oxygen. Mutch pointed out that for simplicity in remembering, the percentages of alveolar oxygen attained are multiples of fifteen, as follows:

Normal	15
Nasal catheters	30
Tent (usual)	45
Tent (extreme)	60
B.L.B. mask	90

It is to be noted that nasal catheters supply a ration of oxygen and the supply is not at all dependent on the demand. In fact, as the demand increases, the percentage of alveolar oxygen decreases. On the other hand, both the oxygen tent and the B.L.B. mask enable the supply of oxygen to be regulated to meet the demand rather than limited to a fixed ration. If only a small amount of oxygen is needed, nasal catheters attached to a Tudor-Edwards spectacle frame are of value. In more serious cases tents or masks are indicated.

Adrenal Cortical Extract—While this method of treatment is still largely in the experimental stage, it offers much promise. Rhoads, Wolff and Lee (1941) studied the use of adrenal cortical hormone in the treatment of traumatic shock accompanying burns. They estimated that without adrenal cortical extract, about forty hours were required for recovery by the capillary walls, of their normal state of permeability for proteins. When 5 to 10 cc of eschatin was administered intravenously every six hours, permeability of capillaries for plasma protein was reduced as early as the eighteenth hour. Other authors have advised 5 cc of adrenal cortical extract intravenously or intramuscularly on admission, followed by 2 cc given by the same route every two hours for the first three or four days following the burn.

The results of treatment with adrenal cortical extract are represented in Figs 3, 4, 5. In Fig 5 a comparison with control cases is shown. In the cases represented at the top of this figure only local treatment was given. The plasma volume of two patients (Cases 25 and 26) represented therein, whose burns involved less than 5 per cent of the body surface, returned to normal in forty-eight and seventy-two hours respectively, after the injury. The plasma volume

of two other patients (Cases 12 and 21) also represented in the top graph, somewhat more than 10 per cent of whose body surface was burned, became normal on the fourth and sixth days respectively after the injury. The middle graph (Fig. 5) represents two patients

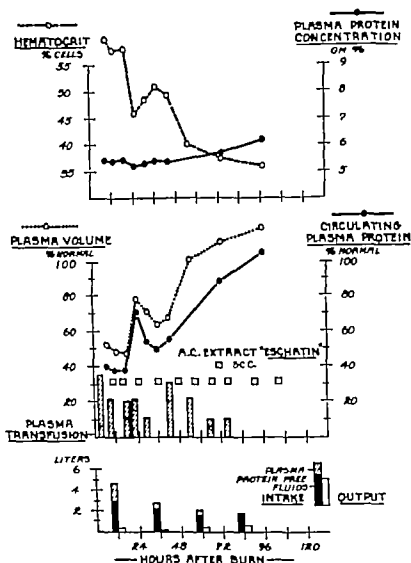


Fig. 3.—Treatment with plasma and adrenal cortical extract in a case of severe burn. Local treatment with gentian violet and silver nitrate also was given. The patient, a Negro girl, aged nine years, recovered promptly from the shock phase but later died with toxic hepatic necrosis (Rhoads, Wolff, and Lee Ann. Surg. June, 1941 J. B. Lippincott Co.)

with extensive burns (15 and 20 per cent of the body surface, mainly third degree) who received adequate transfusions of plasma. The plasma volume was restored to normal in less than fifty hours. The patients represented in the lowermost graph received adequate trans-

fusions of plasma plus adrenal cortical extract; the plasma volume rose rapidly toward normal between the eighteenth and the thirtieth hours. The evidence rendered by this series of cases is merely suggestive and does not prove the efficacy of adrenal cortical extract in controlling loss of plasma in cases of burn.

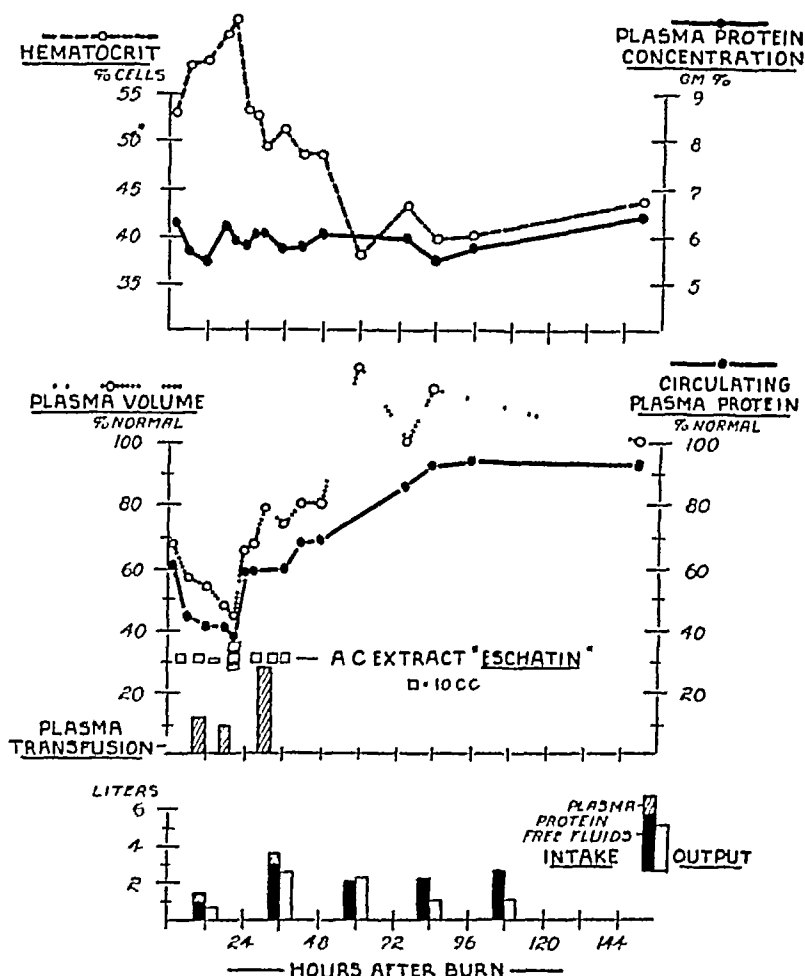


Fig 4—Treatment with plasma and adrenal cortical extract in a case of severe burn. The patient, a white girl, aged twelve years, was treated with gentian violet and silver nitrate locally. The prompt response to general therapy evident in the hematocrit determination and in the determination of plasma volume is represented (Rhoads, Wolff, and Lee. *Ann Surg*, June, 1941. J. B. Lippincott Co.).

Fluid Replacement Therapy.—This is the cornerstone of all treatment of burn shock. Plasma in large amounts is lost, and large amounts of plasma or serum should be replaced. Plasma is generally favored in this country while serum is much used in Great Britain

Solutions of crystalloids such as saline and glucose solutions, have only a temporary effect, as is shown in Fig. 6, *left* and *right*. Acacia has the objection that it forms permanent deposits in the liver. Whole blood is not only harmless, despite the fact that its contained erythrocytes are superfluous, but it is of considerable value in cases in which

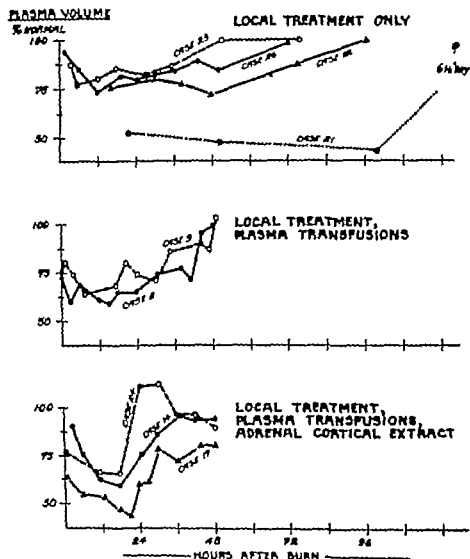


Fig. 3—Comparison of different types of treatment of burns. The case numbers on the face of the graph apply in the article from which the graph was borrowed (Rhoads, Wolff and Lee: *Ann. Surg.*, June, 1941. J. B. Lippincott Co.)

plasma or serum are not available. Volume for volume, however, it is probable that whole blood is not as useful in cases of burns, as is plasma or serum. Plasma and serum have the additional advantage that blood typing is not necessary.

Administration of plasma or serum then, is the best treatment

for burn shock. The substance used should be given intravenously by the drip method. The amount to be given can be calculated in several ways, as is described in the following section.

AMOUNT OF PLASMA—Prompt administration of adequate amounts of plasma controlled by careful and repeated estimation of the concentration of the blood (hematocrit determination, estimation of concentration of hemoglobin, red blood cell count) is of prime importance. There is no more reason always to give a burned pa-

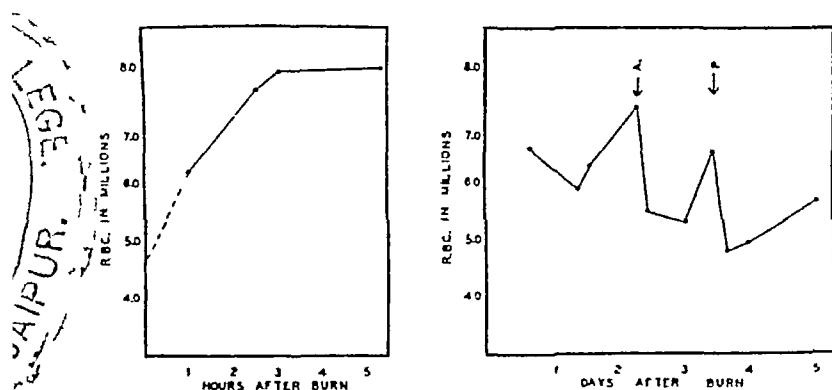


Fig 6—Inadequacy of crystalloid solutions in treatment of burn shock. These graphs represent the red blood-cell counts of two burned patients. The patient represented in the graph to the left, a woman aged twenty-five years, was admitted at 6 P.M. on July 5, 1935, and died six hours later. The patient represented in the graph to the right, a woman aged thirty years, was admitted July 10, 1935, and died six days later. In both cases large amounts of dextrose and saline solution were injected parenterally. Concerning the graph to the left, it is noteworthy that the concentration of the blood was not influenced despite injection of 6000 cc. of dextrose and saline solution in four hours. The graph to the right represents the prompt but temporary effect of 400 cc. of acacia (at A) as well as of 450 cc. of plasma (at P) in lowering the red blood-cell count. Other fluids to the extent of from 6000 to 8000 cc. a day were relatively ineffective. This was one of the first cases of burn ever reported in which plasma was given intravenously but the dosage, as measured by more modern standards, was insufficient (Elman, R. J. A. M. A., January, 1941).

not a pint (500 cc.) of plasma than there is always to give a diabetic patient 10 units of insulin. With blood banks available, it is relatively easy in civilian practice or in military hospitals during periods of relative inaction to obtain large amounts of plasma. The adoption of preserved or dried plasma extends its usefulness to the front line of active engagements. Strumia and associates (1940) gave 7300 cc. of plasma to one burned patient in eleven days. Amounts approaching this have been given by Minot and Blalock (1940) and the Henry Ford Hospital.

In cases of very extensive burn, or when hemoconcentration or other evidence of shock is present, it may be advisable to administer

plasma or whole blood intravenously before local treatment has begun.

There are essentially four methods of calculating the dosage of plasma, as follows

The Method of Harkins (1941).—This is the simplest method and consists in giving 100 cc. of plasma for every point that the hematocrit determination exceeds the normal of 45. If estimations of hemoglobin are used instead of hematocrit determinations, the dosage should be 50 cc. of plasma for every point that the concentration of hemoglobin exceeds the normal of 100 per cent. This dosage applies to adults and involves the assumption that, before the burn was inflicted, the hematocrit determination was normal. A similar assumption is also inherent in the other methods. For children, the amount of plasma is calculated proportionately, according to body weight, with the average adult weight set at about 150 pounds, which is equivalent to almost 70 kg. If the value for plasma protein is less than normal, the method described in this paragraph gives too low a dose. In such a case an additional 25 per cent of the calculated amount of plasma should be added for every gram that the value for protein is below 6 gm. per 100 cc. of plasma. In cases in which treatment is adequate, however, experience has shown that low values for plasma protein are seldom observed in the first few weeks after a burn. Only when blood or plasma has not been given and when the blood has been diluted by intravenous administration of large amounts of crystalloid solutions are low values for plasma protein likely to be observed. Thus, the correction to Harkins' formula for such an eventuality seldom need be considered in actual clinical practice.

The Method of Black (1940).—This method is used in Great Britain and necessitates use of a complicated formula.

The Method of Elkinton, Wolff, and Lee (1940).—This involves use of an even more complicated formula and takes into account both the degree of hemoconcentration and possible low values for plasma proteins.

The First-Aid Method.—When laboratory facilities are not available, plasma, however, may be at hand and some cruder method of estimating its dosage may be advisable. For this purpose the Berkow method of calculating the extent of the burned area (Table 1) is useful. For each 10 per cent of the body surface involved by a deep (blistering) burn, 500 cc. of plasma should be given slowly (that is, 50 cc. of plasma for each 1 per cent area of burn). Another aid in determining the need for plasma is that if the blood pressure is low

TABLE 1—BERKOW'S METHOD FOR ESTIMATING THE EXTENT OF A BURNED AREA

Region	Per Cent of Body Surface Involved
Head	6
Upper extremities	
Both arms and forearms	13 5
Both hands	4 5
Total	18
Trunk	
Anterior surface	20
Posterior surface	18
Total	38
Lower extremities	
Both thighs	19
Both legs	13 6
Both feet	6.3
Total	38 9

or if blood does not flow as easily as is normal from a puncture in the finger or ear, shock is likely to be present to a degree for which plasma will be required.

Comparison of the Methods—An illustrative report of a case is inserted here. It is followed by a demonstration of application of the method of Harkins and the first-aid method for calculation of the dose of plasma.

At 3 A.M., February 11, 1941, the oil-soaked clothing of a man, twenty-one years of age, who weighed 150 pounds (68 kg.), caught fire after explosion of an oil stove. He sustained the burns represented in Figs. 7, 8, 9. The burns involved 43 per cent of the body surface, as calculated by the Berkow formula. On the patient's admission, the hematocrit value was 57, and 300 cc. of plasma was given immediately. This 300 cc. is included in Table 2, from which it can be learned that a total of 2200 cc. of blood plasma was given. This total was administered in the first thirty-six hours following the burn. Resorcinol and tannic acid jelly (Resorcitanol jelly) and sulfaguanidine were applied locally. Desoxycorticosterone acetate (Cortate), 2 cc., was given intramuscularly every four hours for five days and one dose of 1500 units of tetanus antitoxin was administered. In Table 3, the results of certain miscellaneous studies are recorded. The patient was never in a state of severe shock but he died April 7, 1941, fifty-five days after he sustained the burn.

Suppose the calculations of dosage in the case just reported were made according to the *method of Harkins*. The hematocrit determination of 57 on admission is 12 points above the normal of 45 (also

the value for plasma proteins is more than 6 gm. per 100 cc.) Therefore 1200 cc. of plasma is needed.

Again, suppose the calculations were made by the first-aid method. The burns involved 43 per cent of the body surface therefore at least 2150 cc. of plasma is required.



Fig. 7.—A portion of the burns in a case reported in the text. This picture was taken soon after the patient's admission and shows an early tan covered by resorcinol and tannic acid jelly (resorcitannol jelly). The veins on the outer surface of the right ankle, visible through the skin, are early evidence that this is a third-degree burn (C. R. Lam).

It is thus seen that these two methods did not agree in this particular instance.

The actual amount of plasma recorded in Table 2 as having been given on the morning of the day when the burn was inflicted was

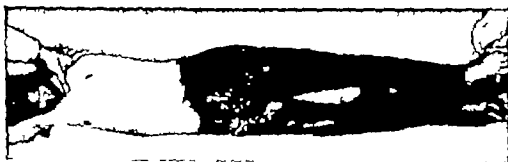
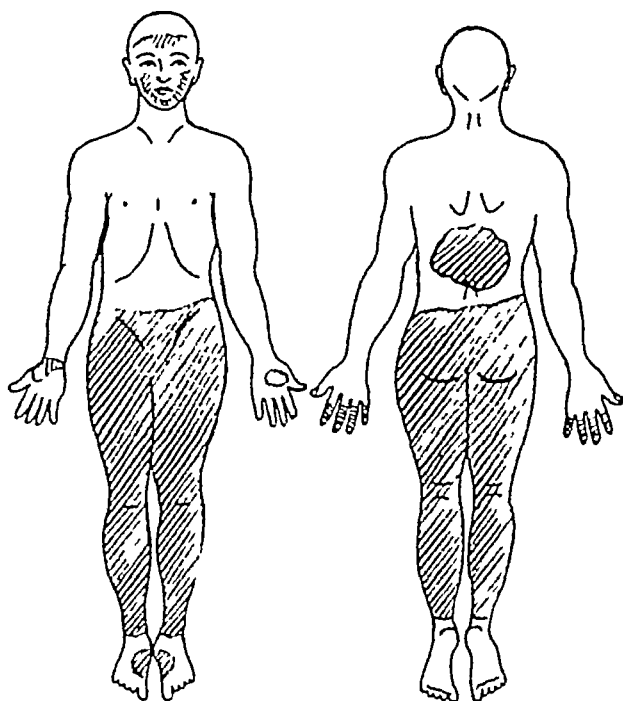


Fig. 8.—Later view of the same patient who is represented in Fig. 7. Complete tanning is now evident.

only 300 cc., which was not as much as any calculations would call for. The validity of the calculations is borne out by the observations made on the afternoon of the patient's admission and recorded in Table 2. Here it is seen that the hematocrit value rose from 57 to

62, rather than falling as it should have done if the initial treatment had been adequate. The results of the application of three methods of calculation, and a comparison with the actual amount of plasma given for each of the days of the first week succeeding the day of the burn is shown in Table 2. During the period of shock,

DIAGRAM OF BURNED AREAS



BURNED AREA ACCORDING TO BERKOW FORMULA.

$\frac{1}{3}$ OF HEAD.....	2 %
$\frac{1}{36}$ OF HANDS AND ARMS.....	$\frac{1}{2}$ %
$\frac{1}{7}$ OF ANTERIOR TRUNK.....	3 %
$\frac{1}{4}$ OF POSTERIOR TRUNK.....	4 $\frac{1}{2}$ %
BOTH THIGHS	19 %
BOTH LEGS	14 %

TOTAL=43% BODY SURFACE

Fig. 9—Outline of burned areas in the case represented in Figs. 7 and 8

when the amount of plasma to be given is an important issue, the calculations agreed closely and the Harkins method stood up well in comparison with the other two more complicated ones. This similarity in the figures applies for the readings obtained on February 11 and 12. Later, after the shock phase had lessened, the readings varied more widely.

TABLE 2.—THE THEORETICALLY DESIRABLE DOSAGE OF PLASMA, CALCULATED BY THREE METHODS, AND THE QUANTITY ACTUALLY GIVEN * AS APPLIED TO THE FIRST WEEK AFTER INFLECTION OF THE BURN

Date, 1941		Feb. 11 (P.M.)	Feb. 11 (P.M.)	Feb. 12	Feb. 13	Feb. 14	Feb. 15	Feb. 17
Hematocrit reading		57	62	56	52	49	40	36
Percentage of hemoglobin (calculated from hematocrit reading)		126	137	124	115	109	89	80
Plasma protein (gm. per 100 cc.)		7.5	7.0	8.1	5.9	6.0	5.9	5.7
Theoretically desirable dosage of plasma in cc., calculated by method of:	1. Eklintou, Wolff and Lee	1250	1760	970	1390	970	20	-450 (excess)
	2. Black	1040	1350	970	650	410	-670 (excess)	-1250 (excess)
	3. Harkins	1200	1700	1100	700	400	0	0
Plasma actually administered,* cc. (after hematocrit and plasma protein determinations)		300	1200	700	0	0	0	0

* The calculations were made from the records of the case a number of months after the case had been encountered. The quantities actually given in this case were based on knowledge available at the time and were then supposed to be adequate.

TABLE 3—SERIAL MISCELLANEOUS STUDIES

Date, 1941	Feb. 11 (A.M.)	Feb. 11 (P.M.)	Feb. 12	Feb. 13	Feb. 14	Feb. 15	Feb. 17
Hemoglobin, gm per 100 cc of whole blood	17.0					12.5	13.0
Nonprotein nitrogen, mg per 100 cc of whole blood	41.0		54.6	54.6	44.4	36.1	38.0
Chlorides, mg per 100 cc. of plasma	347		325	347	330	347	387
Potassium, mg per 100 cc. of serum			17.8				15.2
Sodium, mg. per 100 cc of serum			334				346
Carbon dioxide combining power, volumes per 100 cc. of plasma	16.8		34.9	50.2	65.6	56.0	49.1
White blood cells, per cubic millimeter of whole blood	20,000				22,000	19,000	19,000
Specific gravity of plasma	1.0288	1.0273	1.0305	1.0242	1.0245	1.0242	1.0237

NATIONAL RESEARCH COUNCIL METHOD OF ADMINISTRATION.—

The recommendations of the National Research Council concerning the administration of plasma are, in part, as follows

"The plasma should be given in divided doses. It must never be given by other than the intravenous route. In extremely edematous patients, it may prove impossible to utilize superficial veins. Recourse may be had to the femoral vein. This may be entered at a point $1\frac{1}{2}$ inches (3.8 cm.) below Poupart's ligament. The needle is placed nearly at a right angle to the vein and is inserted to a depth of about 2 inches (5 cm.) If facilities for hematocrit determinations are available, the following rule can be used for guidance regarding the amount of plasma a patient may require for each point that the hematocrit reading is above 50* per cent cells, 100 cc. of plasma should slowly be injected.

"Injections of other fluids, including physiologic solution of sodium chloride and dextrose solutions, should as a rule not exceed the volume of plasma injected in any one twenty-four-hour period. The only exception to this rule is under the condition of severe hemoconcentration (a hematocrit reading above 70 per cent cells) when larger amounts may be injected slowly. The administration of large amounts of neutral sodium salts, such as sodium chloride, will intensify the edema seen in a burned patient. The patient may be permitted to drink moderate amounts of water. No food should be given him until his stomach is retentive and the phase of shock has passed. Then fluids may be taken by mouth as freely as desired."

PREVENTION OF TOXEMIA

When shock is promptly and adequately controlled, as has been outlined in the previous section, few deaths will occur in the first forty-eight hours after a severe burn. Some patients will die later, however including some who, either treated for shock or not, have never shown signs of shock.

Many of the deaths will occur from forty-eight to 120 hours after the burn and are classified as toxic in origin, as is shown in Fig. 1. At necropsy hepatic necrosis frequently is found and parenchymatous changes in the adrenal glands and other organs occasionally are discovered. The clinical appearance of jaundice and relative anuria are the most useful premonitory signs, while an increased icteric index and a decrease in hepatic function serve as helpful evidence. Once severe toxemia occurs, it is usually so fulminating in character that

* Use of the value of 50 rather than 45 per cent seems somewhat confusing, as the normal reading is in the neighborhood of 45

even prompt treatment is of no avail. The chief effort, therefore, should be directed at prevention. Even this is uncertain at times and only a few useful measures are available. These are essentially three.

First, the maintenance of adequate urinary secretion is of importance. Administration of plasma often must be supplemented by intravenous or subcutaneous administration of saline or glucose solution. Since nausea usually is present in these cases, it is fallacious to place much reliance on oral administration of fluids in a case of severe burn.

Second, the hepatic glycogen should be preserved by intravenous administration of adequate quantities of glucose. Fluid containing at least 100 gm of glucose a day is essential, 200 gm. is better and, in cases in which the physician has any suspicion of the presence of hepatic failure, 300 gm should be given. The extremes of the three amounts mentioned correspond to 2000 to 6000 cc. of 5 per cent glucose solution, or 1000 to 3000 cc. of the 10 per cent concentration. When more than 200 gm are needed, part, at least, should be given in the form of the 10 per cent solution, unless there is some other reason for giving a large amount of water, when a more dilute solution may be used.

Third, adrenal cortical extract, as discussed under the treatment of burn shock, also may be of considerable value in preventing toxemia.

The lack of complete efficacy of these measures is evidenced by the fact that occasionally a toxemic death may occur despite them. At present, however, such therapy is the best available protection against this fatal eventuality and should not be neglected. The chloride balance should also be maintained.

PREVENTION AND TREATMENT OF INFECTION

As is shown in Fig. 1, most deaths from burns which occur after the first four or five days are due to some septic complication. Of prime importance in the prevention of such complications is the local treatment of the burned area, discussed in Chapter II of this manual. Without careful aseptic technic in the local treatment, such marked infection may occur that only heroic general treatment will save the patient. The local prophylaxis of infection is of prime importance.

The methods used to control infection are essentially five:

First, general supportive

Second, blood transfusion

as shown

useful

Third, the extensively burned patient should not be allowed to lie too long in one position, despite the difficulty in effecting change of position, lest bed sores or hypostatic pneumonia develop.

Fourth, *tetanus antitoxin* should be administered in all cases in which severe burns are received on the field unless tetanus toxoid previously has been given.

Fifth, treatment with the *sulfonamide drugs* is the chief specific aid. Of these drugs, sulfanilamide and sulfadiazine seem most suited for local application to the burned area, as is discussed in Chapter II of this manual. When such drugs are applied locally, their absorption into the blood stream, as demonstrated by Hooker and Lam, enables them to have a general as well as a local effect. Thus, in cases in which sepsis and nausea coexist, local administration may be the best measure for producing a general effect. In cases of extensive burn, absorption from the burned surface may be so rapid as to cause toxic symptoms unless the administration is quantitatively controlled by frequent determinations of the concentration of the drug in the blood.

In all cases of sepsis from burns, sulfanilamide, sulfathiazole and other drugs of this series may be given orally or parenterally as indicated. In early cases, if urinary suppression is present and sulfathiazole is given, the danger of obstruction of the renal tubules is to be borne in mind. Treatment with sulfonamides lessens the danger of sepsis from burns, but there is as yet no evidence that it can be eliminated entirely.

Dosages of Sulfonamides

Dosages of sulfonamide drugs when given orally in the prevention and treatment of sepsis in cases of burn are much the same as when the drugs are used for other infections. An initial dose of 2.0 gm. of sulfanilamide or sulfathiazole should be followed by 1.0 gm. every four hours day and night for forty-eight hours then 1.0 gm. every six hours day and night for seven days. The initial dose of sulfadiazine should be 2.0 gm., subsequent doses 1.0 gm. every six hours for the first forty-eight hours then 0.5 gm. every six hours day and night for the next seven days. If the patient is not voiding normally (1000 cc. per day) the concentration of sulfadiazine in the blood should be determined daily and the dose should be adjusted downward when a concentration of 10 mg. per 100 cc. is reached. If complete suppression of urine occurs, the drug should be omitted, and fluids forced, orally if possible, and solution of glucose and water given intravenously if necessary. Similar precautions apply to sulfa-

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The methods used to control infection once it has developed are essentially five

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Second, *blood transfusions* are useful when indicated

Third, the extensively burned patient should not be allowed to lie too long in one position, despite the difficulty in effecting change of position, lest bed sores or hypostatic pneumonia develop.

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the hands, and particularly the fingers, are involved unless gloves are worn, in which case only the wrists are burned. Also, the anterior surfaces of the thighs are vulnerable.

On the basis of experience in the present war, surgeons caring for burns in the Royal Air Force have divided the body into five parts, with special directions for the local treatment of each. These areas are (1) face, (2) hands, (3) trunk and extremities, excluding the hands, (4) flexor surfaces and (5) perineum and genitalia

It would be simple if one could be dogmatic and advise the use of one method, for example application of tannic acid, in treatment of all burns. However, it is now evident that it is unwise to try to apply any one method to burns of all degrees in all anatomic situations. Many other factors enter into the choice of local treatment. These include the availability of materials, time and space for certain of the methods, duration of time since injury and so on. Therefore, in this chapter, several of the recognized methods will be described, so that they can be applied whenever indicated. Further experience may change the indications but possibly not the details of procedure of these methods.

COAGULATION METHODS

Tannic Acid Method and Variations

The Davidson tannic acid method of treatment occupies an important historical place in the story of burns. By providing a semblance of rational therapy, it brought interest where there had been little interest before. The method was widely accepted and many early reports indicated a reduced mortality rate coincident with the adoption of tannic acid. It must be pointed out, however, that in the past few years several articles have appeared which suggested strongly that tannic acid might have some adverse side-effects. Several students of the subject of burns who were originally enthusiastic about the Davidson method have recently reversed their positions and now look on tannic acid with distrust. Among these is W. C. Wilson of Great Britain, who in a recent personal communication to the senior author stated that the use of tannic acid jelly (Tannafax) had been discontinued as a first-aid measure in the forward areas of the front in Africa. He reported that conditions in the Middle East are unsuitable for the proper conduct, especially the after-care, of coagulation treatment, and that if coagulation treatment is used, tannic acid should not be included as a coagulant.

Hepatic necrosis is certainly a factor in the syndrome known as

TABLE 4.—STEP BY STEP OUTLINE OF TREATMENT OF BURNS WITH TANNIC ACID-SILVER NITRATE

A. In the emergency room

1. Treat pain and shock with morphine, $\frac{1}{4}$ to $\frac{1}{2}$ grain (0.016 to 0.032 gm.)
2. Give 500 cc. plasma if shock is severe.
3. Take initial hematocrit reading if possible.

B. Admit to burn room

1. Remove and cut away clothing.
2. Transfer patient to burn bed which is covered by a sterile sheet. If possible place mask over patient's nose and mouth.
3. One member of team follows course of pulse, blood pressure, hematocrit reading and clinical signs. If shock is not severe, local treatment is begun.
4. One, or preferably two, men scrub and wear sterile gowns, gloves and masks.
5. Gentle débridement is accomplished by opening blisters and trimming away loose skin. Dirt is removed with saline solution and grease is taken off with ether or ether soap.
6. The burned area on the trunk and proximal extremities is then sprayed with freshly prepared 10 per cent tannic acid solution, followed immediately by a spray of 10 per cent silver nitrate solution.
7. The genitalia, perianal region, hands and face are best treated with another of the local remedies listed in table 5, such as saline dressings, sulfonamide drugs, etc.
8. The patient is then covered with a heat cradle, the temperature within which is not greater than 85° F., and with sterile sheets.

C. Treatment of shock

1. Plasma is administered according to a dosage of 100 cc. for every point the hematocrit reading exceeds the normal of 45. The patient's course and reaction to plasma therapy are followed by repeated hematocrit readings.
2. Oxygen is given in severe cases.
3. Adrenal cortical extract, 5 cc. the first hour and 2 cc. every 2 hours thereafter for 2 to 3 days is given as indicated.

D. Subsequent treatment

1. Infection is controlled with sulfonamide drugs.
2. Transfusions are given to control anemia.
3. Early skin grafting is done to prevent contractures.

"burn toxemia," which appears on the third to the fifth day. It is probably significant that all of a number of papers calling attention to the hepatic lesion have been based on reports of postmortem examination of bodies of patients treated with tannic acid. A partial list of these papers is given: Wilson, MacGregor and Stewart (1938); Belt (1939); McClure (1939); McClure and Lam (1940); Buis and Hartman (1941); and Wells (1942). Wells was the first to publish

an opinion that tannic acid is etiologically related to the changes in the liver

It should be pointed out that hepatic necrosis has not been reported in cases in which the Bettman tannic acid-silver nitrate method of treatment has been used. Here, there is rapid formation of a dry eschar. Immersion in baths of tannic acid solution, and the use of a jelly, may prolong drying unduly and promote the absorption of tannic acid or some decomposition product which is toxic to the liver.

A second objection to tannic acid is that infection is prone to develop beneath the crust. For this reason, it is generally advised that



Fig 10—Untreated burn of the trunk of a child

the material should not be used when the burn is more than twenty-four hours old. Six hours is probably a safer limit. Undoubtedly, if immediate tanning were possible, many organisms would be excluded from the wound

Certain special areas should not be tanned. These are the hands, genitalia, perineum and face, especially if the last is thought to be involved by a third-degree burn in places. The disadvantage in burns of the hands and face is that the eschar prevents early skin grafting. All eschars about the perineum and genitalia become infected sooner or later.

Preparation and Application—The generally accepted method of treating a burn by a coagulation method (tannic acid-silver ni-

trate) will be described in detail, although it is admitted that immediate practical application in actual combat on land or sea would be difficult if not impossible (Table 4)

First, it is essential to remember that a burn is an open surgical wound which is very susceptible to infection. Therefore clothing or first-aid dressings should be left intact until environment and equipment are suitable for carrying out aseptic débridement of the burn. It is desirable for the patient to be transferred to a sterile "burn bed" after the clothing has been cut away. Those who take part in the procedure should wear gowns, gloves, and masks, and others in the room, including the patient, should wear masks. Débridement con



Fig. 11—Eschar resulting from application of tannic acid jelly to the burn illustrated in Fig. 10. For reasons given in the text, the authors no longer advocate this type of treatment for extensive burns.

sists only of opening blisters and cutting away loose pieces of epidermis. This is easily accomplished without anesthesia if adequate sedation with morphine has been obtained. If dirt is present, it is removed by the appropriate solvent and gentle mechanical cleansing. Soap and water are the most useful. In naval warfare, the casualties are often covered with fuel oil. This should be removed by a two-stage procedure. (1) The thick black oil is brought into solution with salt free lard (or Crisco), heavy mineral oil (Nujol) Orvus,* or liquid petrolatum. (2) The mixture resulting from the first procedure is then removed with soap and water.

The area is now ready for tanning. It should be sprayed with a

* Procter and Gamble.

of toxicity and clinical investigation are not available at the time of the printing of this volume. Transportation facilities have been in-



Fig 16—Method of piecemeal removal of the eschar

errupted during the present emergency so that there is a shortage of nutgall tannin and, as a substitute, a tannin preparation known

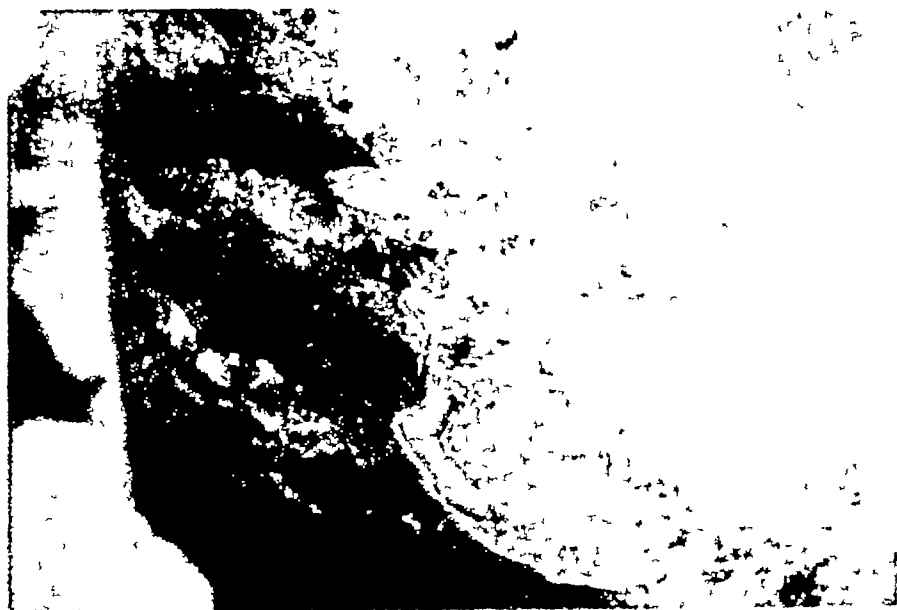


Fig. 17.—An eschar partially cut away

as "quebracho" has been prepared as an ointment. At present it is being investigated clinically. It forms a mahogany-colored eschar

which is more pliable than the usual tannic acid eschar and the substance possesses the advantage that, after it has been applied, it may be covered temporarily with a gauze dressing. Thus, moving the patient is greatly facilitated.

Figs. 18 and 19 show the good result obtained by the tannic acid-silver nitrate method in a case of severe burn of the arms.

The after-care of the eschar consists of attention to the edges until the eschar is ready to be removed, and the ultimate removal of the eschar. Further blisters may form at the periphery of the burn, and these may be opened, débridement performed and further tannic



Fig. 18.—Arms treated by the tannic acid-silver nitrate method of Bettman. Tannic acid jelly applied to face.

acid applied. Several times daily the edges of the eschar are treated with alcohol or other antiseptic substance.

If a second-degree burn is present, the eschar will begin to loosen and curl in ten days to two weeks. As it separates, parts of it are trimmed away piecemeal (Figs. 16, 17). If the burn is of the third degree, a granulating surface remains beneath the eschar. If eschar is still present at the end of three weeks, it is probable that a third degree burn is present and the eschar should be removed. Usually this can be accomplished in short stages without anesthesia or in selected cases, larger areas can be removed with the patient under light anesthesia. The areas then should be prepared for early grafting.

The tannic acid method of treatment has been greatly criticized because of the occasional occurrence of infection beneath the crust. When this occurs, it is a signal for immediate removal of the involved eschar. When pus is present, the eschar is easily cut away. The presence of fever would cause one to search for infection by looking for soft or bulging areas in the crust and by making small windows in areas that arouse suspicion



Fig. 19—Immediate result obtained in the case represented in the preceding illustration

Aniline Dye

This method has been developed by Aldrich and is aimed at the prevention of infection, since the dyes used have a special affinity for the strains of streptococci that frequently are found in infected burns. An eschar is formed, which is more pliable than that formed by the tannic acid method of treatment and its modifications (Figs 20, 21).

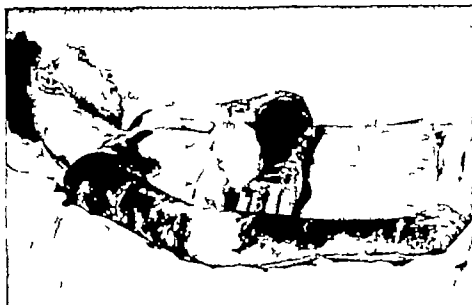


Fig. 20—Type of eschar obtained by repeated applications of gentian violet.



Fig. 21—Result obtained after treatment with gentian violet.

A mixture is made of equal parts of the following dyes:

Brilliant green	1:400
Gentian violet	1:400
Neutral acriflavine	1:1000

This mixture is sprayed on at intervals until a satisfactory eschar has formed. The after-care of the triple-dye eschar is similar to that of the tannic acid coagulum. Areas which become soft early should be painted again, and the edges should be painted daily. A disadvantage of the dye treatment as a measure of first aid is that it obscures the field so that no estimation of the depth of the burn can be made by subsequent observers. Another disadvantage is that the dyes stain bed linens more or less permanently, thus increasing the cost of hospital care.

SALINE SOLUTION

The use of physiologic saline solution in the treatment of burns is the opposite of the coagulation treatment, for here the wounds are kept moist at all times. Many of the recent articles on burns from Great Britain advise the use of saline solution in the primary treatment of second and third degree burns involving the hands, face, flexor surfaces and the perineum. It is also used when complications occur with the coagulation form of treatment and it is desired to remove the eschar and treat the infection.

If only the hands are burned, these are immersed in an arm bath of saline solution at body temperature. Three baths of one hour each are given during the day, the patient being encouraged and urged to move the fingers in the bath. After removal of the hands from the bath, the burns are covered with "tulle gras," which is curtain net with a mesh of 2 mm., impregnated with paraffin. After application of the paraffin netting, moist saline compresses are applied and these are moistened every two hours. They are not allowed to dry out at any time. At the time of the next bath, the dressings are removed down to the paraffin gauze and the extremity is placed in the saline solution. The gauze floats off in the course of the bath. Pieces of necrotic tissue can be washed off by gentle mechanical means.

If the trunk is involved, the entire body can be placed in a saline bath. Physiologic saline solution can be prepared by adding 1 gallon (about 4 liters) of saturated solution of sodium chloride to 32 gallons (about 128 liters) of water. If possible, the patient should remain in the bath for one hour. It is advisable to have some arrangement for keeping a continuous flow through the tub, to remove debris as

it floats off. At the conclusion of such a bath, the burned areas are treated in the manner described above for the upper extremity.

BUNYAN-STANNARD ENVELOPE

This method has been given a reasonable trial in Great Britain and it has been recommended by several authors. The burned areas are enclosed in water proof envelopes of oiled silk, which are sealed to the skin. The envelopes are made in several shapes to fit various parts of the body. There is an opening for the entrance of fluid and another for its exit. When a burned patient is admitted to the hospital, the burn is douched with 5 per cent sodium hypochlorite solution at body temperature. This cleanses and removes charred tissue. Each day the envelope is irrigated three times with 2.5 per cent sodium hypochlorite solution by running the envelope full and leaving it full for twenty minutes. Agitation is carried out during this period, after which the fluid is drained out. The envelope is left in place continuously. If there are extensive burns of the trunk, the patient is placed in a collapsible tub which serves as a bed between irrigations. Bunyan believes that the method is valuable because it removes dead tissue before the latter can give rise to toxemia; also the covering is transparent and healing can be observed. Moreover, free motion is possible throughout the treatment.

CLOSED METHODS

Pressure Dressing

The surgical group at Chicago headed by Dr. Sumner Koch has advised a very simple treatment for burns. This consists of thorough débridement, such as that described for the tannic acid treatment. It is especially urged that those working around the wound be masked. When the wound is clean, it is covered with fine mesh vaseline gauze and pressure is applied by means of fluffed gauze, mechanics' waste and elastic bandages or stockinet. Such a dressing is not disturbed for ten or twelve days. When it is removed, many areas will be found to be healed, while necrotic portions of the skin are ready to be removed by appropriate methods. Skin grafting is performed early.

Cod Liver Oil and Plaster Casts

On the basis of his experience in the Spanish Civil War, Trueta has advocated the extensive use of plaster immobilization in the treatment of various types of wounds. Such a procedure may be indicated in certain cases of neglected burns. Débridement is done

A mixture is made of equal parts of the following dyes. . . .

Brilliant green	1.400
Gentian violet	1.400
Neutral acriflavine	1:1000

This mixture is sprayed on at intervals until a satisfactory eschar has formed. The after-care of the triple-dye eschar is similar to that of the tannic acid coagulum. Areas which become soft early should be painted again, and the edges should be painted daily. A disadvantage of the dye treatment as a measure of first aid is that it obscures the field so that no estimation of the depth of the burn can be made by subsequent observers. Another disadvantage is that the dyes stain bed linens more or less permanently, thus increasing the cost of hospital care.

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under general anesthesia, after which the burned areas are covered with vaseline gauze and plaster is applied to the involved parts in the usual manner. This provides immobilization and prevents pain. Suppuration may take place under the cast and a bad odor may develop but this would not necessarily indicate a change of dressing. The casts would be removed and the injured areas inspected when it was felt that necrotic tissue had sloughed and granulating portions were ready for grafting. The Germans have used cod liver oil and plaster casts extensively. The chief exponent of this method is Löhr, who treated 1777 patients in eight years.

LOCAL TREATMENT WITH SULFONAMIDE COMPOUNDS

Pickrell, of the Johns Hopkins Hospital, recently has described his experience with a solution consisting of 3.5 per cent *sulfadiazine* (2-sulfanilamido-pyrimidine) in 6 per cent triethenolamine. This solution is sprayed on with an atomizer. It can be applied by a nurse while the surgeon is scrubbing to do the débridement, which is carried out in the same manner as in the tannic acid treatment. After the débridement, the patient is placed on the sterile burn bed and is covered with a cradle. The burns are sprayed every hour during the first day, every two hours during the second day, every third hour during the third day, and every four hours during the fourth day. A thin, transparent eschar forms by this time. The healing process can be observed under this eschar. It is said that this eschar is pliable enough to permit some motion so that contractures are prevented. The removal of the eschar begins in ten days. This is facilitated by compresses of the sulfadiazine solution or a sterile mineral oil spray followed by physiologic saline compresses.

The concentration of the drug in the blood should be determined daily.

Sulfadiazine and other sulfonamides are being placed in ointments, jellies and emulsions. The usual concentration of drug is 5 per cent. At the time of the writing of this article these materials are being extensively investigated and it is probable that they will soon be generally available.

CHOICE OF TREATMENT

The foregoing methods of treatment have been described without indicating clearly on what types of burns they should be used. This vagueness is necessary because of the disagreement which exists at present. If a physician who is treating burned patients under-

stands the method of application of the foregoing procedures, he can easily adapt his treatment to meet changes in opinions or instructions as these arise. A tentative outline of indications for the various forms of treatment is given in Table 5

TABLE 5.—CHOICE OF LOCAL TREATMENT FOR APPLICATION TO VARIOUS BURNED REGIONS OF THE BODY

Portion of Body	Treatment for Burns of	
	First and Second Degree	Second and Third Degree
Trunk	Tannic acid-silver nitrate* Tannic acid Sulfadiazine spray Pressure dressing (Koch) Triple dyes	Tannic acid-silver nitrate* Tannic acid Pressure dressing Saline tube Triple dyes
Face	Sulfadiazine emulsion Vaseline dressing Saline packs Triple dyes Tannic acid jelly	Sulfadiazine emulsion* Vaseline dressing Saline packs
Hands	Pressure dressing* Saline baths Triple dyes	Pressure dressing* Saline baths
Flexor surfaces	Saline packs Triple dyes Sulfonamide pastes Tanning methods	Saline packs Sulfonamide pastes Tanning methods
Perineum and genitalia	Sulfadiazine emulsion* Boric ointment* Saline baths Triple dyes	Sulfadiazine emulsion Boric ointment Saline baths

The treatments indicated by asterisks were recommended by the Conference on Burns of the National Research Council on January 7 1942. These recommendations were presented at the Panel Discussions on Burns of the American College of Surgeons at various cities in March 1942.

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CHAPTER III

1

CARE OF GRANULATING SURFACES, THE EARLY PLASTIC TREATMENT OF BURNS

ROY D. McCLURE, M.D.
and
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ONCE the tannic acid or other eschar has been removed, the burn becomes a granulating surface. Since this is essentially a continuous process, often the physician who has been responsible for the early care of a burn must be sufficiently versed in plastic surgery to complete effectively the healing of the granulating surface. Furthermore, haste is essential since the longer a granulating surface remains, the more likely are several complications to ensue.

POSSIBLE COMPLICATIONS

Contractures

These develop as long as the burned surface remains unhealed and this may occur even when splints, casts or other forms of restraint are used.

Infection and Debilitation

These are obvious dangers.

Carcinoma

This late complication, while rare, practically never develops in a rapidly healed burn no matter how extensive. It is the result of neglect.

Poor Subsequent Healing and Keloid

The longer a burned surface remains unhealed, the thicker the scar tissue becomes and the greater the menace of a poor cosmetic result. In Great Britain it has been found that practically all deep burns of the dorsum of the hand will require a skin graft either primarily or secondarily that is, on the granulating surface or later to release contractures.

THE IMPORTANCE OF PROMPT HEALING

For these reasons, and others too apparent to mention, prompt healing of a burned surface should be the prime aim of treatment once the stages that must be passed through on admission of the patient are over. Early in the treatment of a burned patient, the saving of life is of chief importance; later, when his life is safe, the functional and cosmetic results arouse chief interest. Because treatments with these two aims merge, they must often be carried out not only by the same physician, but in the same hospital or unit. The responsibility of the physician for an acute burn does not end until the surface has healed. Once the surface has become epithelized, the need for haste is over and subsequent reparative measures, such as application of flaps and alterative plastic operations may be instituted at any time. These measures constitute the late plastic treatment of burns and are thus more or less a separate subject.

The early plastic treatment of burns consists, therefore, of such care of the granulating surface as will hasten or effect epithelization of the defect

SPONTANEOUS HEALING

A certain amount of spontaneous healing of all defects takes place. Healing may be hastened by general and local measures.

General Measures

Adequate Nutrition—A diet high in sulfur (eggs), vitamin C and proteins is advisable. Edema of the nutritional type should be controlled by forced or orojejunal feeding or by transfusions of blood or plasma.

Prevention of Anemia—It has been said that it is harder for skin or skin grafts to grow when the concentration of the patient's hemoglobin is below 70 per cent than for grass to grow on the Sahara desert. The remedy, blood transfusion, is obvious.

Control of Infection—In certain cases, use of the sulfonamide drugs controls infection.

Local Measures

The reader will notice that some substances and methods recommended for use in the next few paragraphs also have been mentioned in Chapter II. However, whereas Chapter II deals with immediate care, Chapter III deals with treatment at a somewhat later time. To outline the complete treatment in both chapters seemed desirable.

Moist Dressings and Soaks.—These are the chief bulwarks in the modern care of granulating surfaces and can be applied in several ways. *Tub baths* are useful when available and have been favored by Brown and Blair. Five per cent saline solution in such baths may be painful on the first immersion, but the patient soon becomes accustomed to it. In Great Britain a gallon (about 4 liters) a minute of physiologic saline solution is run through the tubs used for treatment of burned patients. When only an arm or hand is burned, local bathing in a small tub made to fit the arm is especially useful. A galvanized iron trough can be made to place under a burned leg. Then saline solution or other reagents can be dripped on the leg from suspended flasks and the excess fluid carried off by the trough to a collecting waste basin. Physical therapeutic measures applied in the bath are of especial importance. If the hands are burned, special attention should be paid to the interosseus and lumbrical muscles and, in general, active movements are better than passive ones. In the later care of the burned hand, warm paraffin baths may be useful. Saline compresses are especially useful on the face and, when used there, a jacomet bib assists in keeping the patient and the bed dry. The comfort of the patient in the tub is often of great assistance in preserving his morale and this is of extreme importance in rehabilitating patients who have sustained war burns.

The use of saline baths in treatment of burned patients recently has found considerable favor for wartime use in Great Britain. Mowlem (1940) advised their use and stated that after the first one or two immersions they are painless. O'Loughlen (1941) favored Hebra's water baths. Gillies (1940) Bunyan (1940) Hudson (1941) and Wakeley (1940 and 1941) advised irrigation within one of the Bunyan-Stannard oiled silk envelopes, developed by Bunyan.

If *Bunyan-Stannard envelopes* are employed, the course of healing can be watched through the oiled silk and dead tissue and stale exudates are washed away in the course of irrigation. The use of these envelopes is shown in Figs. 22 to 25. In this method, 2.5 per cent sodium hypochlorite solution is run by gravity, for about twenty minutes three times a day through the envelope. During the interim the wound is left undisturbed. The envelope prevents evaporation and keeps the granulating surface in a moist atmosphere such as has been advised by Smith, Risk and Beck (1939). All of this is accomplished without resorting to the use of moist dressings, the changing of which is painful and produces bleeding.

Cod Liver Oil Ointment.—This substance has been found to have a slight bactericidal effect, to be one of the few actual stimulants



Fig 22—Severe third-degree burn of right hand treated with Bunyan envelope This photograph shows the granulating surface several weeks after the burn and just before grafting Primary treatment consisted in pressure saline dressings supplemented by cod liver oil and later by saline irrigations inside Bunyan envelope

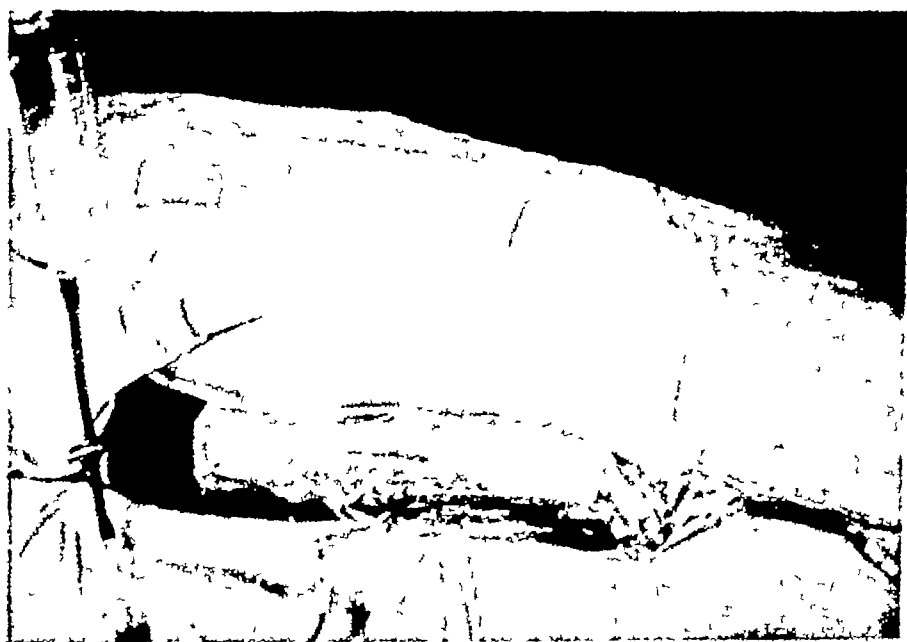


Fig 23—Use of Bunyan envelope The burned hand shown in Fig 22 is encased in the envelope, through which saline solution from the vessel in the upper left hand corner of the picture is run The fluid then issues from the tube near the finger tips

of epithelial proliferation and at the same time to be free of any deleterious irritating action.

Vaseline Gauze, Xeroform Ointment Gauze Tulle Gras and Scarlet Red or Oxyquinoline Sulfate Scarlet R Ointment Gauze—All of the substances named have their place in the treatment of granulating surfaces resulting from burns. Tulle gras is a close meshed curtain net impregnated with soft paraffin (98 parts), balsam of Peru (1 part) and olive oil (1 part) with sometimes the addition



Fig. 24—Result of saline irrigations followed by use of machine graft. This photograph was taken three weeks after the first grafting.

of 0.1 per cent oil soluble percaine* base. Tulle gras is especially in favor in Great Britain for use on burns and skin graft donor sites.

The formula of the oxyquinoline sulfate scarlet R ointment is as follows

	gm. or cc.
Oxyquinoline sulfate (chinosol)	0.6
Trichlorbutanol (chlorotone)	2.4
Scarlet R, Biebrich's medicinal	6.0
Oleum ricini (castor oil) q.s. ad	120.0

* Percaine, now called "nupercaine" is a proprietary local anesthetic agent. It is the hydrochloride of alpha-butylloxycinchonic acid-gamma-diethylenedi amide.

Gauze is impregnated with the finished ointment and is used as is vaseline gauze, best in conjunction with saline dressings.

Sulfonamide Compounds—In cases of infection of the granulations, continuation of use of sulfonamides locally, as described in Chapter II, up to almost the time of grafting, may be advisable in conjunction with oral administration.

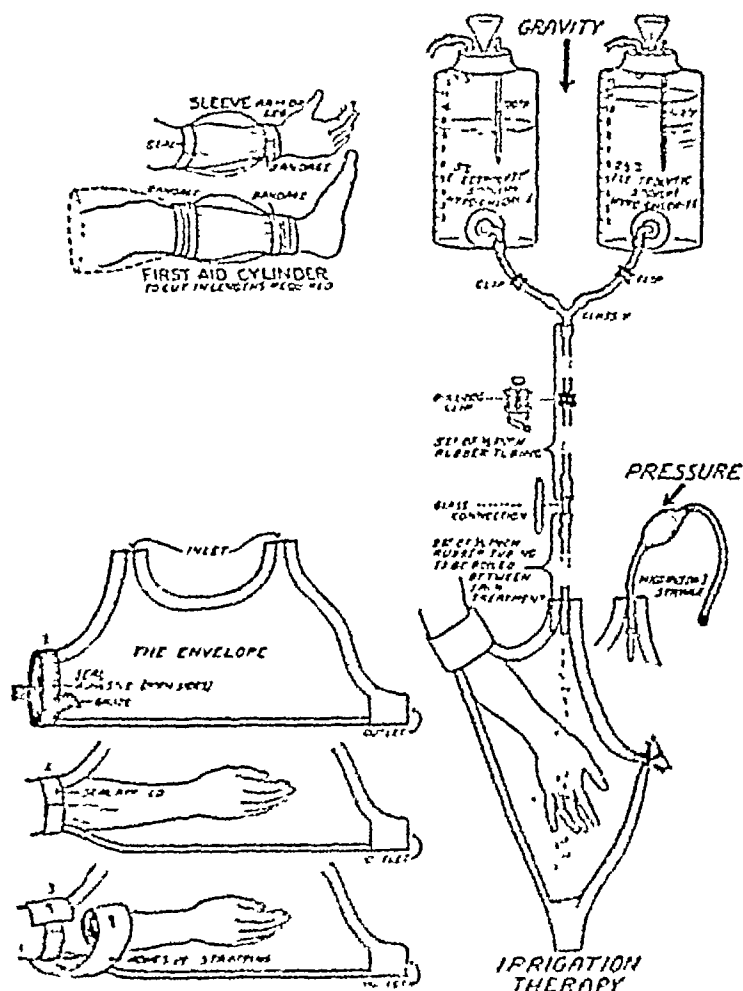


Fig 25—Use of Bunyan envelopes (after Hudson, Brit. M J, July 5, 1941)

SKIN GRAFTING

Skin always should be grafted when passive measures fail or when the size of the granulating surface indicates that the natural process will at best take too long. *Almost invariably the error is made of waiting too long before grafting skin and of not using skin grafts in enough cases* Seldom does the opposite mistake occur. A good arbitrary rule is to apply grafts to every granulating surface which

is more than four weeks old, which is more than 5 cm. in diameter or which looks as if it would take more than three additional weeks to heal spontaneously. Seldom are there exceptions to this precept.

There are essentially three kinds of grafts to apply to granulating surfaces: (1) small, deep grafts, (2) thick, split grafts, and (3) "machine" grafts. The use of full thickness or pedicle grafts at this stage is too risky and should be avoided. The surface should first be epithelized and made sterile and then these more complicated, deeper grafts can be applied in secondary operations if necessary.

The indications for the three main types of graft are given in succeeding paragraphs.

Small, Deep Grafts

These grafts, introduced by Reverdin (1869) and improved by Davis (1914) are the simplest to cut; the only instruments necessary are a needle, a scalpel and a syringe of local anesthetic agent. They take where most other grafts fail and, if a few are lost, no great damage is done. They are especially useful over extensor surfaces. An advantage of them is that they can be obtained from concave donor surfaces; this advantage also applies to machine grafts but not to thick split grafts. In Chapter IV (pp. 62-64) exactly how this graft differs from the Reverdin (pinch) graft is explained.

Thick, Split Grafts

These have much the same field of usefulness as the small, deep graft but are more quickly obtained. A 3 by 8 inch (about 7.5 by 20 cm.) graft is cut in a minute by those facile with the razor or skin grafting knife. The cosmetic result is much better than with pinch grafts and, if the same donor site is well cared for, it can be used as many as three times.

Machine Grafts

These are taken with the Padgett Hood dermatome. The use of this machine is shown in Figs. 26, 27. The general technic used is similar to that of taking thick, split grafts in so far as preparation of donor and recipient sites, transfer of graft, dressings and after-care are concerned. The only difference is in the taking of the graft. The adhesive qualities of the roller allow the graft to be taken from almost any surface, whether concave or convex and this gives a decided advantage in cases of burn, in which it is not always possible to pick and choose the donor site. The machine insures against cutting into the fat and a graft of even thickness is almost universally

obtained. The machine method has the special advantage for war-time that it enables relatively inexperienced surgeons to cut as good



Fig 26—Taking of machine graft from left thigh. The graft is seen on the drum of the instrument and the area from which the graft was cut is seen as a multitude of fine bleeding points



Fig. 27—Application of machine grafts. The grafts are sutured to a granulating area on the inside of the left calf in a case of third-degree burn

grafts as can be obtained with the razor or Blair-Brown knife only after years of specialized practice. The exigencies of warfare require

that specially trained general surgeons be ready to do wholesale skin grafting on short notice. One of the authors of this chapter has used machine grafts more than forty times in the past two years for covering granulating surfaces due to burns and is convinced of their value for this purpose.

The Technic, Step by Step

Instructions for the technic of skin grafting in the early plastic treatment of granulating surfaces following burns occupy the remainder of this chapter

1 *Preparation of Donor Site.*—These sites should be remote enough from the granulating surface to prevent infection, should be on unexposed portions of the body, and, if possible, the right lower quadrant of the abdomen and any sites of possible future pedicle grafts should be avoided. In general, the thighs, abdomen, and back are the best areas. For small, deep or for thick, split grafts any type of preparation of the skin is satisfactory and the donor area can be outlined with a 5 per cent alcoholic solution of brilliant green. This serves the double purpose of acting as a guide for local anesthesia and to mark the upper surface of the grafts. When machine grafts are to be used the skin is cleansed with ether followed by alcohol, then dried and painted with the adhesive glue* which is allowed to dry for about sixty seconds, after which the graft is immediately taken.

2 *Anesthesia.*—Local infiltration with 0.5 per cent procaine solution is advisable when possible, especially if patients are anemic, as it cuts down loss of blood. It is useful for the taking of small, deep grafts. General anesthesia may be preferable for the other two types of graft since the grafts usually are sewed to the recipient area and this process itself requires a general or block type of anesthesia.

3 *Removal of Grafts.*—To remove a small, deep graft, the skin is punctured and held up by a needle wedged back as far as possible between the jaws of a small, straight hemostat. The graft is then cut toward the surgeon, with a scalpel to which is given a sawing motion. The grafts are cut in rows, leaving a space of about 0.5 cm. between each two grafts and making the grafts themselves about 0.7 cm. in diameter. Because blood may drip down a slanting surface, the lowermost rows are best cut first. After a few rows have been cut, a warm, moist sponge is applied to control bleeding.

For thick, split grafts, an ordinary straight razor can be used or better the long, sharp knife designed by Blair and Brown. Similarly

* This glue is furnished with the Pedgett Hood dermatome.

boards or flat dishes covered with gauze can be used to hold the skin taut, but better results can be obtained with Blair and Brown's suction retractors. The skin is covered with a thin layer of vaseline and the cutting is done with a sawing motion, the movement coming from the shoulder and the cutting being done toward the surgeon. It has been advised to cut deep enough to include from half to three-fourths of the skin. More practical advice is to cut as deep as possible without going into fat. Once the fat has been cut into, a new graft should be started. In ideal cases the donor surface is a field of multiple small bleeding points.

When *machine grafts* are used, both the skin and surface of the drum are painted with the glue which is allowed to dry for about sixty seconds. If a graft smaller than the full size of the drum is required, it is much better to apply the glue to the entire drum and then wipe off the unneeded portion with a piece of dry sterile gauze than to attempt to spread the wet glue in any sort of pattern. Similarly, when the drum is to be used several times at the same operation, dry gauze is the best means of cleaning it between grafts. In all cases the last centimeter of length of the drum should be wiped free of glue as this obviates the use of a scalpel in cutting the graft loose from the patient. The drum, with the knife set to cut at a thickness of about 0.008 to 0.012 inch (0.2 to 0.3 mm.) and with the axle and outer surface of the knifeblade holder greased with a small amount of sterile vaseline, is held in the left hand and the knife in the right. A thickness of 0.012 inch (0.3 mm.)* is standard for male adults of military age. Persons more than sixty years of age, or children from ten to sixteen need 0.010 inch (0.25 mm.) whereas younger children furnish the best grafts when the knife is set at a depth of 0.008 to 0.010 inch (0.2 to 0.25 mm.). When dermatome grafts are applied to sterile surfaces, especially as used in the late plastic cure of burns, they may be thicker than those indicated for covering granulating surfaces. Two practical points are to press the upper end of the drum very firmly against the skin where the cutting is to begin, as a good start is essential, and not to allow assistants to retract the skin as this merely pulls it away from the drum and is harmful. By gradually turning the drum with the left hand and cutting backward and forward with the right, a graft the size of the surface of the drum, and of uniform thickness, is quickly obtained in a majority of the cases.

4. *Dressing of the Donor Site*—The warm, moist sponges are re-

* Assuming that each gradation on the dermatome scale is 0.002 inch (0.05 mm.). Each dermatome may have to be individually calibrated by clinical results.

moved and the area is washed with fresh, sterile physiologic saline solution. Silver foil or xeroform ointment gauze is then applied and covered with a dry dressing which is sealed with adhesive tape. Optional methods for treating the donor site include the production of a tannic-acid eschar thereon, or dusting sulfanilamide powder gently and evenly over the surface, followed by application of a layer of vaseline gauze. This latter method of use of sulfanilamide powder has proved especially successful. The dressing is not changed before the fourteenth day unless a marked odor is present. By this time, in about three-fourths of the cases healing will be complete. When an early change of dressing is required, only the outer layers should be removed. If small, deep grafts have been taken such healing will be by third intention, whereas, if thick split, or machine grafts are properly used, it will be by modified first intention.

5 Transfer of Grafts to Granulating Surface.—The habit of transferring grafts to the infected, granulating surface, and then returning the soiled instruments to the sterile donor region is a surgical error. With thick split or machine grafts, the taking of the graft should be completed before the recipient area is touched. When small, deep grafts are used, the simplest and best technic is to place the grafts on towels that are kept moist with warm saline solution and then, later to transfer them to the granulating surface.

6 Preparation of Granulating Surface.—The clinical appearance of granulations is of more value in deciding the time for grafting than is the bacterial count. Granulations suitable for grafting should be firm, clean, pink, and not exuberant or edematous. Edema can best be controlled by several days of pressure dressings (see Chapter II) and by avoidance of a dependent position. Strong antiseptic substances should be avoided and, while the surrounding skin may be painted with an antiseptic agent, saline solution or ether is sufficient for use on the granulations themselves. In certain instances the granulations should be removed, but in each case the decision should be made on individual merits. Removal necessitates use of general anesthesia. In general, granulations less than three months of age need not be removed and removal is more often done for thick, split grafts and for machine grafts than for small, deep grafts.

7 Application of Grafts.—Small, deep grafts are placed about 0.5 cm. apart and are firmly pressed onto the granulations, epidermal side upward. Thick split and machine grafts are applied identically. Small fenestrations to permit escape of serum, and suturing around the edges and across the surface of the graft are advisable with the latter two types of graft. In some cases, when the graft is applied to in

fectured granulations, it may be advisable to dust sulfanilamide powder over these granulations before applying the graft. If this is done, the powder should be evenly distributed and not more than 1 gm. of powder should be used for each 10 square inches (about 65 sq cm.) of surface. Pressure is very important in dressing the area as the powder tends to promote the formation of serum.

8 Dressing Grafted Area—This is a step of far more than apparent importance and should be done as meticulously as the operation itself. The grafts are carefully covered with xeroform ointment gauze or tulle gras and this, in turn, is covered with several layers of dry gauze. The flat surface of a bisected rubber sponge or sea sponge is then applied and fastened in place with a gauze bandage. In place of the sponges, sterile mechanics' cotton waste is useful. The gauze bandage is then covered with elastic, adhesive tape which anchors the dressing to the surrounding skin and if possible a splint or cast is applied. Too much pressure is almost worse than too little, but adequate immobilization should be continued for several days. The fate of a skin graft is sealed during the first forty-eight hours. If marked infection is present, Dakin's tubes may be incorporated in the dressings. A good routine to follow in such cases is to irrigate through the tubes twice daily with 2 quarts (about 2 liters) of water, followed by 2 ounces (60 cc) of a saturated solution containing one of the sulfonamide drugs, such as sulfadiazine.

9. Changing the Dressing—This should be done on the third to eighth day, depending on the amount of infection present at the time of grafting. When the change is decided on, it is best to soak the dressing for several hours beforehand to prevent sticking. In certain cases, the xeroform gauze or silver foil may be left on to be removed at the second dressing. If thick split or machine grafts have been applied, easily removed stitches are taken out at the first complete dressing and others at later times.

10 After-Care.—Except for the extra need of gentleness until the grafts have become firmly established, the after-care is much the same as that of a granulating surface. If the first grafting is not successful, a second should be attempted.

FOR PARTICULAR EMPHASIS

The immediate responsibility of the surgeon in a case of third-degree burn does not cease until the granulating surface has entirely healed. The importance of early skin grafting for third-degree burns cannot be emphasized too often. The mistake of grafting "too little and too late" is far more frequent than the reverse.

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CHAPTER IV

SKIN GRAFTING FOR CONTRACTURES FOLLOWING BURNS*

JAMES BARRETT BROWN M.D

LOSS OF FULL THICKNESS OF THE SKIN†

WHEN the full thickness of the skin is destroyed by a burn over a large area, an open wound results and, unless the lost skin is replaced early, healing will occur by contracture of adjacent tissue and by "scar" epithelium, or permanent healing may never occur.

Almost any treatment that promises as little work as possible is likely to become accepted but, so far no application has been found that will produce normal skin and, when it is stated that a certain preparation will insure complete healing without scarring, it apparently is not realized how healing occurs in the presence of loss of superficial portions and of the full thickness, of the skin.

Tendency to Spontaneous Healing

This heading is reminiscent of one which appears in Chapter III and, in the present chapter there is a certain amount of intentional recapitulation in order that some desirably instructive illustrations may be utilized.

There are marked individual variations in the epithelization of these wounds 1. An occasional patient may present healing without deformity after loss of a large portion of the full thickness of the skin. 2. In other cases healing may be permanent, but with various degrees of contracture and deformity 3. In some cases epi-

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†The illustrations and their legends form part of the text to avoid repetition and may be referred to directly as they are listed in the text.

many times not sufficient to give the permanent surface necessary for the area. The scar epithelium is dry and excessive keratotic growth may form. The epithelium is subject to cracks and irritation and may break down over large areas on slight trauma or circulatory disturbance.

The reason for this unsatisfactory healing can be clearly shown microscopically. The scar epithelium lies on a fibrous tissue base, the fibers of which usually seem to be arranged parallel with the surface. This fibrous tissue may be of excessive thickness and may have a very poor blood supply. The scar epithelium that has regenerated over the area has not normal derma to support it and attach it to the subcutaneous tissues. The cells have extended out over the base in almost a straight line so that no, or very few, papillae are present. The epithelium itself is devoid of hair and sebaceous glands; it may be but a few layers of cells thick in one place and, close by, there may be marked hyperkeratosis (Fig. 29).

Pain and Contractures

The foregoing possibilities have been concerned with the epithelial healing, but the center, or granulating surface itself, and the underlying functioning parts also should be considered. When a large wound has been open over a long period, pain usually becomes most severe, presumably from more nerve endings developing in the bed; this forms a bad cycle for any proper care of the wound and is one of the underlying causes of morbidity and death in the presence of old, unhealed burns, since both the patient and the attendants may become unable to cope with the situation (Fig. 28, D).

The second consideration relative to the whole area is contracture and deformity of the surface, tendons and joints. Displacement and fixation due to contracture of skin and surface, as the wound pulls in toward the center, may be called primary contracture. All wounds exhibit this type of healing and the more lax the area is, the easier the healing with the least apparent deformity, but widespread losses will soon lead to primary contracture and disability (Fig. 30).

Secondary contractures can be considered those that affect underlying tendons which, though not damaged directly, have been held contracted so long, both voluntarily and later by the overlying scar, that they are actually shortened.

Fixation of joints may occur, especially in the hand, and subluxation of the knee and other large joints may take place.

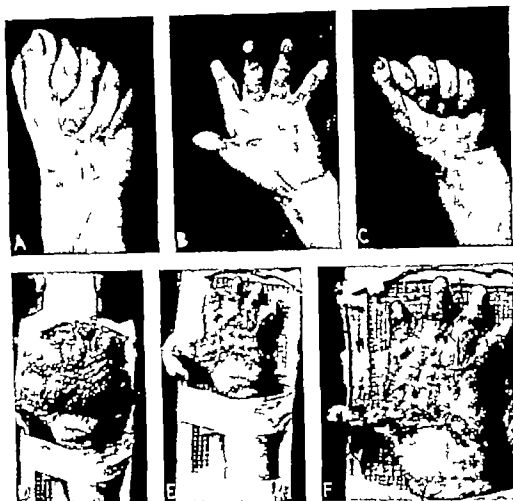


Fig. 30.—A, Complete flexion deformity and webbing of the fingers several years after a burn. B and C, Full function obtained with one full-thickness graft and two subsequent procedures to relieve the webbing. D Hand shown at time of first dressing, with molded marine sponge in place. Dorsal surface well padded. E, Grease gauze of fine mesh next to the graft which is dry indicating full take of the graft. F Full take shown with edge sutures; individual mattress sutures through the surface; stab holes through the graft; and fingers held extended with silver wires through each tip and attached to the wire splint. Stab holes usually are omitted (author's article in *Ann. Surg.*, Vol. 107 J. B. Lippincott Co.)

Prevention of Deformity, Care of Raw Areas, Objects of Treatment

In this section late deformities are the primary consideration, but for the prevention of these deformities the following summary of the care of open wounds, as discussed in Chapter III, is included.

The local care of open wounds has for its object the cleaning of the areas as quickly as possible, so that the lost surface can be restored with skin grafts before contractures have occurred and before debilitation and pain have developed beyond control. Clean-

ing with soap and water, and surgical drainage accompanied by saline dressings or saline baths, usually suffice, but use of antiseptic agents, Dakin's solution, or sulfanilamide may be thought necessary.

A fundamental of dressing all raw wounds is that old linen, or gauze of very fine mesh be placed next to the surface so that granulations will not grow up through the coarse meshes of overlying gauze, stick to the gauze and cause pain. When cellulitis has been controlled, grease dressings on fine gauze or linen can be used and the dressings are not changed as often as is necessary for wet dressings.

A firm pressure or elastic dressing may be of great advantage, and discolored, edematous granulations may be flattened out into a firm, bright red surface without use of chemicals or without cutting them away.

The continual saline bath may have the important result that most secondary contractures will be straightened out by the voluntary effort of the patient, without the use of traction or restraint. Many patients are extremely grateful for the bath and realize their first comfort in it, and it occasionally has been a lifesaving measure. If the bath causes any unfavorable reaction, it is omitted.

It seems impossible to sterilize large, open areas, but careful evaluation of the general condition of the patient and of the gross appearance of the granulations and surrounding tissues usually suffices for the determination of the time for operation, many deep burns are ready for grafting in three weeks' time.

TYPES OF FREE SKIN GRAFTS FOR MAKING REPAIRS

A consideration of the histology and thickness of the skin is of some importance in relation to the various types of free grafts.

The Reverdin or Pinch Graft

To take skin for this type of graft the cut is shallow, passing just through the epithelium. The surface area of the graft is 0.5 sq cm or less. It happens to be the least useful of all the types of skin graft, usually thicker, or deeper, sections of skin give much better results. This thicker graft has been reported on extensively by Davis who has suggested the term, *small, deep graft*. It will grow in many fields where others will not, but oftentimes, because of this fact, little attention is paid to the preoperative preparation of the wound, with the result that many of these grafts are lost. Their greatest value is in areas that are covered by clothing, as they give a rather spotty appearance, they are also of service at times for hastening healing in areas where the surface cannot be made clean enough for other

Ollier-Thiersch Grafts

These usually are thought to contain only the epidermis, but they are cut in sheets in contrast to the small bits taken as pinch grafts. In reality, even the thinnest Ollier-Thiersch graft usually includes a thin layer of derma. These grafts are too thin to be of much use in making repairs in large areas where there must be a firm surface (Fig 32, A)

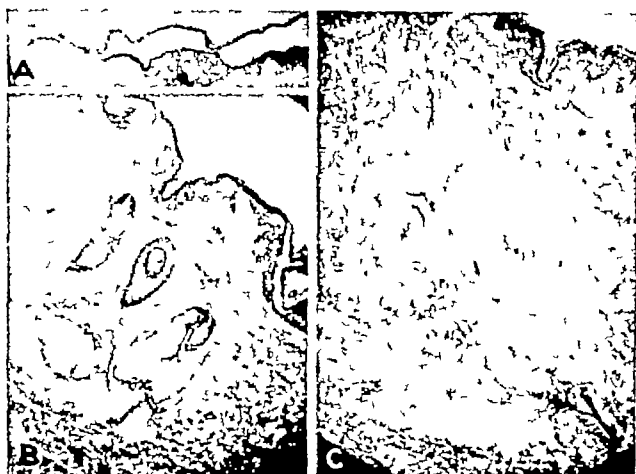


Fig 32—Thickness of skin grafts All three grafts cut from the same area of a single patient and photographed under the same magnification A, Average thickness of Ollier-Thiersch graft. Most texts state that the skin is taken through the papillary layer, but even the thinnest ones usually include some derma. B, A thick split, or thick Ollier-Thiersch graft. The thickness can be graduated up to three-fourths of the full thickness C, Full-thickness graft (author's article in *Internat. Abst. Surg*, Vol 67)

Thick, Split Grafts

After a surgeon has tried to use the thin, Ollier-Thiersch graft, he almost automatically will cut deeper. A graft of from half to three-fourths of the thickness of the whole skin is perhaps the most useful in making all repairs of raw surfaces. This graft could be designated as a "thick Ollier-Thiersch" or as a "thick split graft" (Fig 32 B)

Healing of Donor Sites—Where large surfaces are to be covered it is necessary to obtain large pieces of these grafts without cutting entirely through the derma so that healing of the donor site can occur rapidly from the epithelial glands that are left behind in the lower layers of the derma. Rapid healing of the donor area is most important as it makes large amounts of skin available at one time. One hundred square inches (about 645 sq. cm) are frequently taken, and as high as 221 square inches (about 1,425 sq. cm) have been transferred in one operation.

The deep glands in the derma "dedifferentiate" into squamous epithelium and cover the surface in from six to eight days, in from twelve to twenty days no dressing is necessary. This dedifferentia-



Fig. 33.—Healing of donor site. A, Biopsy from donor site of split graft after two days, showing deep glands and uncovered surface. B, Biopsy from patient five days later. There is complete surface coverage with squamous epithelium that has "dedifferentiated" from the deep glandular epithelium. The connection from the deep gland to the surface can be seen in one area (author's article in Internat. Abstr. Surg., Vol. 67)

tion can be shown clearly in microscopic sections and, if healing is uninterrupted by infection, successive "crops" of skin can be cut from the same area, as much as four crops have been taken from

one area. There is a marked variation among patients in regeneration of the surface, it is usually several weeks before a second graft



Fig 34—A, B, and C, Application of a waste dressing over a split graft, D, E, and F, dressing of the donor site of a split graft. The deep grease gauze is left in place twelve days, G, and H, final fixation of the waste with gauze rolls and of the joint with plaster of paris (Brown, Byars, and McDowell Arch Surg., Vol 40)

can be taken, but such a graft has been obtained as early as nineteen days after the previous crop (Fig. 33)

The donor sites are dressed carefully with greased gauze of fine mesh and the dressing is allowed to remain from ten to fourteen days. Healing occurs most promptly when there is little or no activity (Fig. 34)

The Cutting of Thick, Split Grafts.—The most essential equipment is a very sharp, long knife of the amputation variety. With this long knife, large grafts can be cut rapidly and, the larger they are the more easily they can be applied. Grafts up to 18 by 5 inches (about 46 by 13 cm.) can be obtained from suitable thighs. With

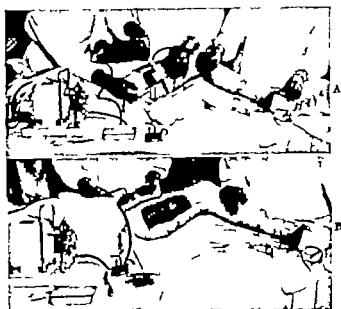


Fig. 35.—A, Technic of cutting graft. The skin is lightly greased with vaseline; the knife is very sharp and about 18 cm. long. Strong suction is supplied by the pump and delivered to the suction retractor through the tube. The assistant makes countertraction with the soap dish. B, Grafts as large as necessary or as large as the thigh will supply are taken. The graft is shown spread out on the table and should always be clamped immediately to the cover to prevent loss (Brown and Blair: Surg., Gynec. and Obst., Vol. 60)

the suction retractor described in 1929 or by elevation of the skin with large tenaculums, fairly large grafts can be cut even from the abdomen (Fig. 35)

The dermatome of Padgett or Hood seems to be successfully used by surgeons who are not accustomed to the freehand cutting of grafts. It should be used guardedly to prevent ruining the donor site. There are several other instruments for cutting grafts, any of which may be used.

Many methods of cutting split grafts have been described and different terms have been applied to the grafts. If the full thick

ness of the skin is not taken, then the skin necessarily has been split in two and the graft is at least anatomically a "split" graft. To have a new type of skin graft would require a new type of skin but, as new authors realize how utilizable the split graft really is, enthusiastic reports are made, in which new terminology is used

The Application of Thick, Split Grafts, Pressure Dressings.—These grafts are applied to the area after granulations have been carefully and smoothly shaved off, after any healed contractures have been fully opened by dissection, or after scar tissue has been excised. They are held firmly in place with running triple zero silk sutures all around and multiple mattress sutures over the surface. Stab holes may be made through the grafts to provide for drainage if there is much contamination, but otherwise they are omitted.

If the area is fairly free from contamination, a pressure dressing is applied with a layer of grease gauze over the graft. More flat gauze is applied over this and then, as a medium of pressure, cotton mechanics' waste is spread over the whole area and bound firmly on with heavy gauze rolls and plenty of adhesive tape. Neighboring joints should be fixed with crinoline or plaster, if necessary. The fixation of this pressure dressing is as important as any step in the operation (Fig 34). Other mediums of pressure can be used, such as marine sponges and waste rubber, but it is important that the final pressure be created by the outlying bandage.

Postoperative Care of Split Grafts—Extreme care should be taken with the dressings which are done, first, from the third to the sixth day, the edges of the graft are trimmed away, sutures are removed, and some mild antiseptic agent is painted over the area. If there is not much cellulitis, a fine mesh, grease gauze dressing can be used but, if infection is present, a wet dressing should remain in place for twenty-four hours or sulfanilamide, in powder or solution, can be used.

These grafts will take in almost 100 per cent of instances under proper conditions, and do not need a great deal of postoperative care. For these reasons they are used in all places possible, rather than full thickness grafts, and also because much larger amounts of skin are available.

Preparation of Cotton Mechanics' Waste and Marine Sponges for Pressure Dressings

Two types of waste have proved satisfactory for most dressings. The material is cheap, easily handled and much more easily incorporated in a dressing than are marine sponges (Figs 34, 36).

The waste is obtained in bales of various size.* The white variety is of shorter fiber and is bleached. The coarser material is unbleached and has longer threads, so that it is used for larger dressings. The cost was 12 cents per pound when this was written (1942)

The bale is autoclaved at 30 pounds (13.6 kg.) of pressure for one hour; then it is opened, and suitable amounts are resterilized in drums, jars or wrapped packages for handling at the time of operation.



Fig. 36.—A, Two kinds of mechanical waste used as a medium of pressure in surgical dressings. B, Use of coarse waste to hold a graft on the neck; sutures from the edges are tied over the surface after one gauze sponge has been used to cover the waste (Brown, Byars, and McDowell Arch. Surg., Vol. 40)

Marine sponges should be of good quality, large, soft and "wool-form," and should be bleached. Bleaching does not injure them, does not cost any more and helps in general in handling and in having the dressing appear clean. These sponges are prepared by beating out the loose dirt, washing in soap and water and then soaking in

* Wiping Materials, Inc., 2028 North Main Street, St. Louis, or some similar firm.

1:1000 mercury bichloride or mercury cyanide solution for forty-eight hours. They are then washed out in saline solution, allowed to dry out and are put away. They should not be stored wet but can be kept in drums or sterile wrappers. They are, of course, moistened again to soften them when they are applied. The sponges are not to be handled by the clean nurse and are not to be put directly on wounds, because their sterility is only relative.

Preparation of Grease Gauze of Fine Mesh

Old linen was originally used to prevent sticking but bandage gauze of fine mesh will suffice and is easier to handle. It is routinely applied to all open wounds and to all grafts. It is best prepared from bandage 4 inches (10 cm) wide and can be laid flat in pans, as is the ordinary vaseline gauze, or it can be kept in a full roller for easy application to extremities. It seems that plain vaseline is likely to cause maceration but that the addition of almost any of the recommended medicaments will tend to prevent this.

Innumerable drugs have been recommended for this use but the following are usually relied on: merthiolate 1:1000, xeroform 4 per cent, scarlet red 5 per cent and sulfanilamide. Sensitivity to drugs, of course, has to be taken into account.

Homografts and Delayed Grafts

By using autogenous split grafts wherever possible, and preserving the donor areas fairly well, it is possible to find enough skin to make acceptable repairs in most cases.

Fresh homografts are employed occasionally when it is thought that the patient cannot stand a long operative procedure and when there is no sign of spontaneous epithelization. Homografts usually will take satisfactorily but are absorbed in a few weeks. However, the few days' respite that the patient receives while these grafts are in place may actually be a turning point in his recovery and spontaneous epithelization may increase. Homografts might be thought of as emergency dressings for intractable wounds but they have no place in repairing late deformities (Fig. 37).

It is hoped that some method of getting homografts to persist may be developed; this is one of the most important things that could be accomplished in reconstructive surgery. Work on the blood groups has not disclosed any relationship to the persistence of the grafts up to the present, although more investigation on the subgroups should be done. Homografts from identical twins have been shown to grow and persist so that they could be used if a twin ever needed skin from someone else.

It is possible to use successfully autografts that have been stored for several days, but this fact is not very important clinically, especially with regard to split grafts, for they can be cut rapidly and add but little time to the operation.

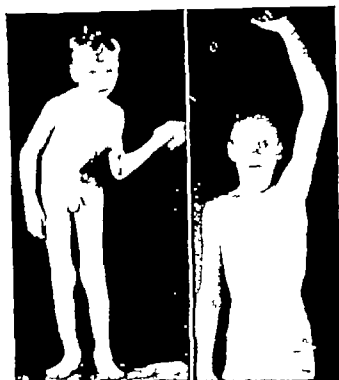


Fig. 37—Very deep, widespread burn. Patient tided over critical period with large, split homografts from mother. Axilla then released in two operations in which split grafts were applied. Generalized shortening of skin across abdomen and flank released two years later with split grafts. Normal function recorded a second time, nine years after operation. Grafted area extends from iliac crest all the way up through axilla and down over arm (Brown and McDowell: Surg., Gynec. and Obst., Vol. 72)

Full-Thickness Skin Grafts

Full-thickness grafts (Fig. 32, C) are not put over large, raw surfaces because the size required is prohibitive, and the take of a full thickness, dissected graft is not as certain in contaminated fields as that of the split graft. The full-thickness graft is used to correct healed deformities, when a clean operation can be done. Its main application is to surfaces not covered by clothing and to those where healing seems to give a better chance of immediate return of the area to normal if such grafts are used. Even when these grafts are employed, pigmentation may be troublesome and require the use of cosmetics.

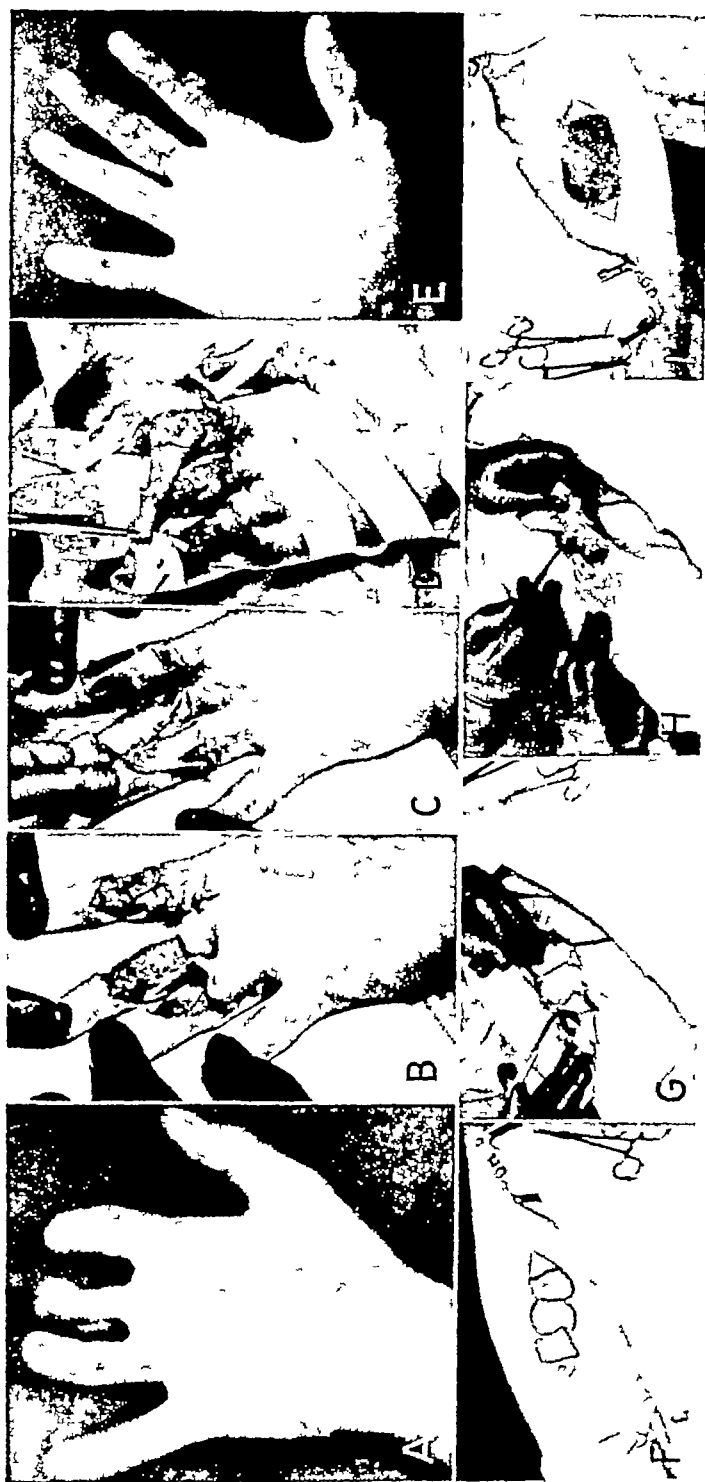


Fig 38—A, Flexion deformity of fourteen years' standing B, Careful opening without damage to nerves or tendons, careful hemostasis, fingers can be straightened C, The interdigital spaces have been made out of the contracted palmar skin Lead patterns have been cut of all three defects D, The graft is sewed accurately all around the edges of the separate defects, and mattress sutures are put through the surface if necessary E, Result three months later. Patient satisfied with function, which is apparently normal F, The patterns have been traced and outlined through the skin of the inner part of the thigh G and H, The full-thickness of the skin is raised with as little damage as possible, using fine forceps and gauze for holding No fat is taken up on the graft I, The graft is free and the resultant wound can be sutured or covered with a split graft from an adjacent area (author's article in Ann. Surg., Vol 107. J. B. Lippincott Co)

Details, Preparation of Bed for the Graft—The hand shown in Fig 38 can be taken as a model for the details of applying a full-thickness skin graft. The fourteen-year-old contracture, represented

in Fig. 38, A, gave promise of being susceptible of fairly good correction because movement of joints and activity of tendons had been preserved. That lengthening of such tendons by traction will be possible is not certain until the area has been opened but, for a successful correction in one operation, it is necessary that the entire deformity be opened and the hand maintained in complete correction during the period of healing of the graft.

Fig. 38, B, represents the freeing obtained by removal of the small, transverse, heavy scars and then careful dissection backward of the palmar flaps without damage to the nerves or complete exposure of the tendons. The edges of the defect are not straight lines but have "darts" in them to allow maximal replacement of skin. If a straight, narrow graft is put in a child's finger, and a keloid should develop along the edge deformity might result. The palmar flaps are utilized in making new interdigital spaces and for part of the coverage of the finger; then patterns of the defects are cut in thin sheet lead. (Fig. 38, C) Hemostasis is carefully effected with pressure and ligatures of white, triple zero silk.

Cutting the Graft.—The patterns are outlined on the inner side of the thigh and incision is made just through the skin (Fig. 38, F). The full thickness of the skin is then elevated, using small forceps or gauze traction on the graft and some countertraction on the edges of the donor area to prevent any subcutaneous fat from adhering to the graft. Some small particles may persist and can be removed with scissors later; if these would retain their viability they would be of great advantage (Fig. 38, G and H). The resultant defect of the donor area now can be closed directly or if it is too large, it can be covered rapidly with a split skin graft from an adjacent area (Fig. 38, I).

Application of Graft.—The hand is fastened to a wire or aluminum splint with sterile adhesive tape, with the fingers extended, then it can be handled easily. The graft is sewed accurately into place all around the edges, in order to favor obtaining primary union, where edges are too loose to allow normal stretching of the graft, reefing sutures may be taken to pull the edges back away from the defect. The tension with which the graft is sewed on—that is, from side to side—is best described as normal skin tension. It is not pulled tightly like a drumhead, or left so loose that it may wrinkle. If the graft is larger, then it is held down to the bed with additional mattress sutures through the surface (Fig. 38, D). The result is represented in Fig. 38, E.

Dressing the Graft.—One layer of greased gauze of fine mesh is

placed, very smoothly, next to the graft. Over this one or two gauze flats are laced and cotton mechanics' waste is packed carefully between the fingers. Over this more waste is placed and bandaged



Fig 39—Extreme deformity of neck and jaw corrected in two operations with thick split grafts and one operation with a full-thickness graft. Recorded after a period of growth of eight years, with complete restoration of contour. Surface smoothness can be improved with further grafts when patient wishes. Both axillae also repaired with thick split grafts (Brown and McDowell: Surg., Gynec. and Obst., Vol 72)

firmly in place. A board splint is added for stability and the ends of the fingers are left visible. Fig. 30, D, E and F represent a hand at the time of the first dressing, which was done eight days after

operation. The graft had fully taken and there was no sign of infection, but it was necessary to maintain dressings for two weeks longer. Wet applications would be used in such a case if inflammation should occur; otherwise use of the greased gauze would be continued. The sutures could be removed at the first or later dressings, and there might be an apparent delay in healing because so much of the surrounding, heavily keratinized skin might come away.

The splint is usually maintained in use for three weeks and then movements are gradually restored. This long fixation, which is primarily for the graft, helps toward lengthening of tendons also, but it does not do joint movement any good. If there is a tendency for contraction after this period the patient is usually allowed to go about his activities during the day and at night the dorsal splint is reapplied. Either an aluminum splint or a simple one made of wood is held on with adhesive tape.

REPAIR OF LATE DEFORMITIES

In the repair of late contractures from burns, free skin grafts can be used extensively and they give permanent bearing surfaces in many instances. They often can be substituted for tedious, laborious use of pedicled flaps that require multiple operations.

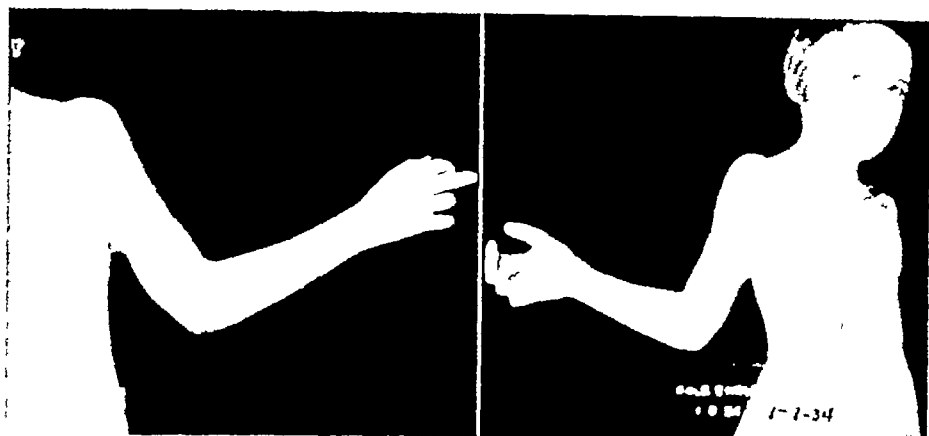
If sinuses extend down into scar folds and harbor micro-organisms that are detrimental to the taking of a skin graft, preliminary opening is made, and sometimes a huge open wound is produced. In the grafting of all contaminated open wounds, the thick split graft is used rather than a full-thickness graft because of the greater assurance of its taking in such a field (Fig. 39).

If the late contraction is healed so that a clean operation can be done, then the use of a full-thickness graft can be undertaken much more safely; but even here, in many instances, the split graft can be used satisfactorily and may even be required if the area to be grafted is so large that the necessary amount of skin of full thickness cannot be sacrificed (Figs. 40-41).

Z-Plastic Operations

In repairing healed contractures all surrounding skin that it is possible to utilize is, of course, put to use. If there is dense scarring and marked contracture, seldom can flaps be turned in from the surrounding area; the entire mobilization is one of dissection and release of binding scars. In old cases, in which the scar has been drawn out into a web and there is little or no deformity occasionally

it is possible to effect the repair by using the web itself. This is commonly called a Z or reversed Z-plastic operation; however, the openings seldom actually resemble a Z and the main point to re



A



B



C

Fig 40—A, Growth of arm to side from loss of wide area of skin over arm, thorax, axilla, and both axillary folds B, Fixation of dressing after application of thick, split grafts A quilted bed pad is incorporated over the sponge pressure dressing and held with gauze rolls and adhesive tape C, First dressing after operation, showing the sea sponges as they have become molded in place (Brown and Blair. Surg., Gynec and Obst., Vol 60)

member is that the two surfaces of the web are saved and fitted across each other to obliterate the web. In many cases adjacent flaps, or the two surfaces of the web, can be combined with free skin grafts

to cover remaining open areas. Scar flaps should not be too large because their blood supply may be extremely uncertain (Fig. 40)



Fig. 40.—(Continued.) D Complete release of the arm has been obtained and a full take of the thick split grafts is shown, one operation. Incision extends from anterior superior spine of ilium, over flank, thorax, axilla, and half way down arm. Although none of the scarred surface was removed, it can be seen that the only skin flaps available for closing were small ones over the flank. The triangular insert of skin over the arm is to be noted as important in relaxation. E, Complete function apparent, but close observation shows a very tight, heavy scar over hip that makes a general tightness and some hindrance to activity. F The lack of sufficient skin surface has been corrected in one operation; this has been accomplished by simply opening the scar and dissecting the edges back without sacrificing any surface, and then covering the defect with thick split grafts, as shown (Brown and Blair: Surg., Gynec. and Obst., Vol. 60)

Generalized Shortening of Skin

This is a condition in which there is no evident distortion but definite lack of flexibility and of ease of function may exist. This may result from failure to release the scar sufficiently or from growth of the area without enough enlargement of original grafts. The condition may be likened to clothes that are too tight or it may be said that the skin envelope is too small. This means that scar and its contracture persist until released, if completely released early in life, the repair should last; if, with increased growth, the shorten



on from two operations in which split grafts were
though nerve was exposed in dissection Patient
a time after a nine-year period of
and Obst., Vol 72)



Fig. 42.—Marked distortion and jaw twisted down into open bite; corrected in two operations in which split grafts were used and two operations in which full-thickness grafts were applied. Normal profile and occlusion of teeth resulting and recorded after a period of growth of ten years (Brown and McDowell: Surg., Gynec. and Obst., Vol. 72)



Fig 41 — Complete function from two operations in which split grafts were applied No footdrop even though nerve was exposed in dissection Patient came in for amputation Recorded a second time after a nine-year period of growth (Brown and McDowell Surg, Gynec and Obst, Vol 72)



Fig. 42.—Marked distortion and jaw twisted down into open bite: corrected in two operations in which split grafts were used and two operations in which full-thickness grafts were applied. Normal profile and occlusion of teeth resulting and recorded after a period of growth of ten years (Brown and McDowell. *Surg. Gynec. and Obst.*, Vol. 72)

ing does appear, then more skin can be let in by simply opening the tightest area, allowing the edges to retract and filling the rest of the defect with suitable grafts. Where the scar tissue is very thick, however, the edges will not retract when opened and then large flaps have to be excised down to good, soft tissue and still larger flaps applied.

REPAIR OF SPECIAL AREAS

In repair of areas in which the opening is of long standing the condition is complicated by primary and secondary contracture. It is often safest simply to apply grafts to the open areas first, then to open the contractures. This is to obtain healing, even in a deformed position, so that the surface can be clean enough later to permit a deeper opening into the region of contracture, without the spread of infection. Thus, entire correction of any deformity is effected. When the deep opening is made, the new, clean areas can be covered completely with grafts. If any dirty sinus exists, they should be opened widely as a preliminary step, so that they can be cleaned the same as any other raw area (Fig 42).

Hands

Prevention of deformity of the hands is so important that special note on early care is included. When the hands are burned, great effort should be put forth to prevent the deep infection that so rapidly cause fixation of tendons and joints and will produce deformities that may never be overcome. The first treatment should be cleansing with soap and water and gentle débridement, then the hand should be wrapped in greased gauze of fine mesh and bandaged. Daily soaking in saline solution from a half hour to an hour, with new dressing with further débridement can then be carried out until the wound is ready for grafting. If there is no cellulitis, a good, grease dressing may be left in place for several days. This method might be called "surgical drainage" in contradistinction to the dressing of the areas with tannic acid or plaster of paris. Active movement should be encouraged while the soaking is taking place. The fingers should be dressed apart and the entire hand should be kept in the position of function. The average burn should be ready for grafting in three weeks, if tendons have not been exposed, and frequently the single application of a split graft may be all that is necessary. If there has been an extensive, deep burn, it is often advisable to "dress" the wound with a thick split graft as soon as the sloughs and tendons have been separated and the granulations are clean, so

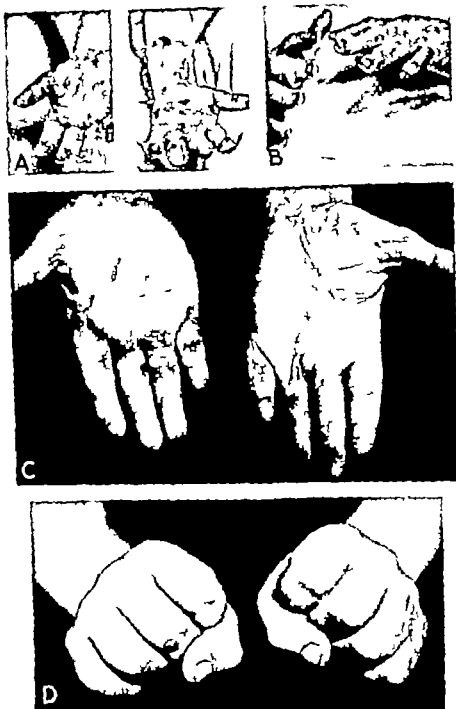


Fig. 43.—A and B, Fresh burns of both hands from falling in fire. Cared for as described in text and ready for grafting in twelve days. C and D Result of one grafting operation shown five years later. Both hands were "dressed" in split grafts; that is, a large graft was put over the entire area and sewed accurately in place. There was growth of the graft over the raw parts and it was trimmed away where the surface regenerated. Patient was cared for by Drs. McDowell and Guinn, Surgical Service at Barnes Hospital (Illustrations from author's article in *Internat. Abstr. Surg.*, Vol. 67)

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REPAIR OF SPECIAL AREAS

In repair of areas in which the opening is of long standing and the condition is complicated by primary and secondary contractures, it is often safest simply to apply grafts to the open areas first, without opening the contractures. This is to obtain healing, even in a deformed position, so that the surface can be clean enough later to permit a deeper opening into the region of contracture, without fear of the spread of infection. Thus, entire correction of any deformity is effected. When the deep opening is made, the new, clean, raw areas can be covered completely with grafts. If any dirty sinus tracts exist, they should be opened widely as a preliminary step, so that they can be cleaned the same as any other raw area (Fig 42)

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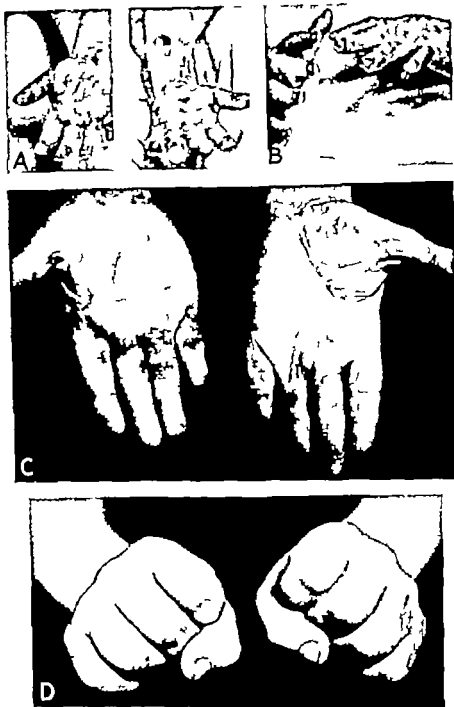


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healing may stimulate activity and joint fixation may be limited, later, deeper repair can be done

In "dressing" the areas with grafts, many cases will be encountered in which regeneration of the surface will take place anyway. The graft is put over the entire area, however, for assurance that the raw areas will be covered, later the graft can be cut away from the healed surfaces (Figs. 43, 44).

Late Deformities Repaired with Free, Thick Split Grafts—Many late deformities can be repaired by carefully dissecting the scar, so

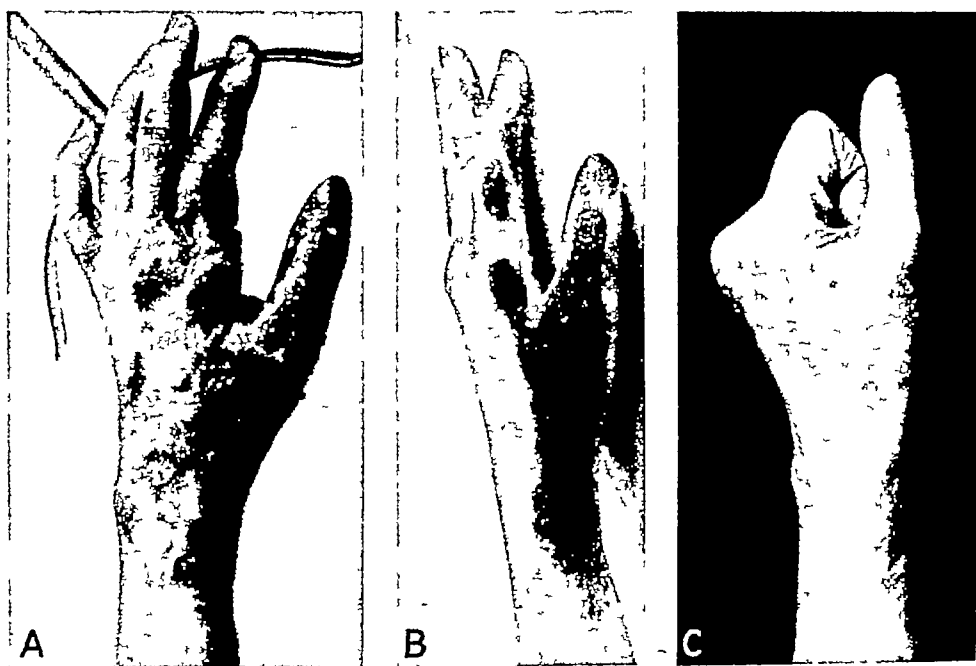


Fig 44—A, One month after injury in laundry mangle and burn B, Two days later, after gentle débridement and use of saline dressings Operation performed three days later, at which the granulations were carefully cut away down to a firm base, and the entire area was covered with a single large, thick split graft. C, Complete and permanent function after four years (author's article in *Ann. Surg*, Vol 107 J B Lippincott Co)

that complete correction is obtained with little or no exposure of tendons Then the defects are covered with thick split grafts (Fig. 45).

Partial Restoration of Function—In some cases the deformities are so bad that complete function never can be expected These deformities result from excessive, widespread loss of surface, deep losses of tendons and dislocation of joints from primary or secondary contractures Many of these deformities might be prevented by adopting the view that the lost skin should be replaced early There probably

never can be any excuse for the completely webbed, or "degloved" hand as it has been called.

A patient whose deformity was extreme is represented in Fig. 46, A, B. He could not even dress himself. The thumb, fingers and wrist were all out of functional position and the situation seemed almost hopeless. In four operations, however the scars gradually were removed and the surface was covered with thick split grafts, so that the thumb was brought fairly well around into position and the position of the wrist was restored (Fig. 46 C, D) Complete function was not restored but the patient was able to get about normally and some of his work in occupational therapy is illustrated in Fig. 46, E. He was also able to draw and he studied art. His left hand



Fig. 45—A, Widespread deep burns of the dorsums of both hands corrected by careful dissection of the scar without damage to the tendons and covering each area with one large split graft. B and C, Complete permanent function three years later (author's article in *Ann. Surg.*, Vol. 107 J. B. Lippincott Co.)

was in worse condition than the right and was repaired in the same way

Healed Deformities Repaired with Free Full thickness Grafts.—

A lost dorsal surface in many instances can be replaced successfully with thick split grafts (Fig. 45) Although there seems to be no actual measure of advantage of the full-thickness graft, as far as final function is concerned, it is often relied on in cases where widespread, clean dissection and removal of the binding scar can be accomplished.

The patient represented in Fig. 47, A, had completely lost use of the hand, the thumb and wrist were entirely out of the position of function and the fingers were so held back that metacarpophalangeal

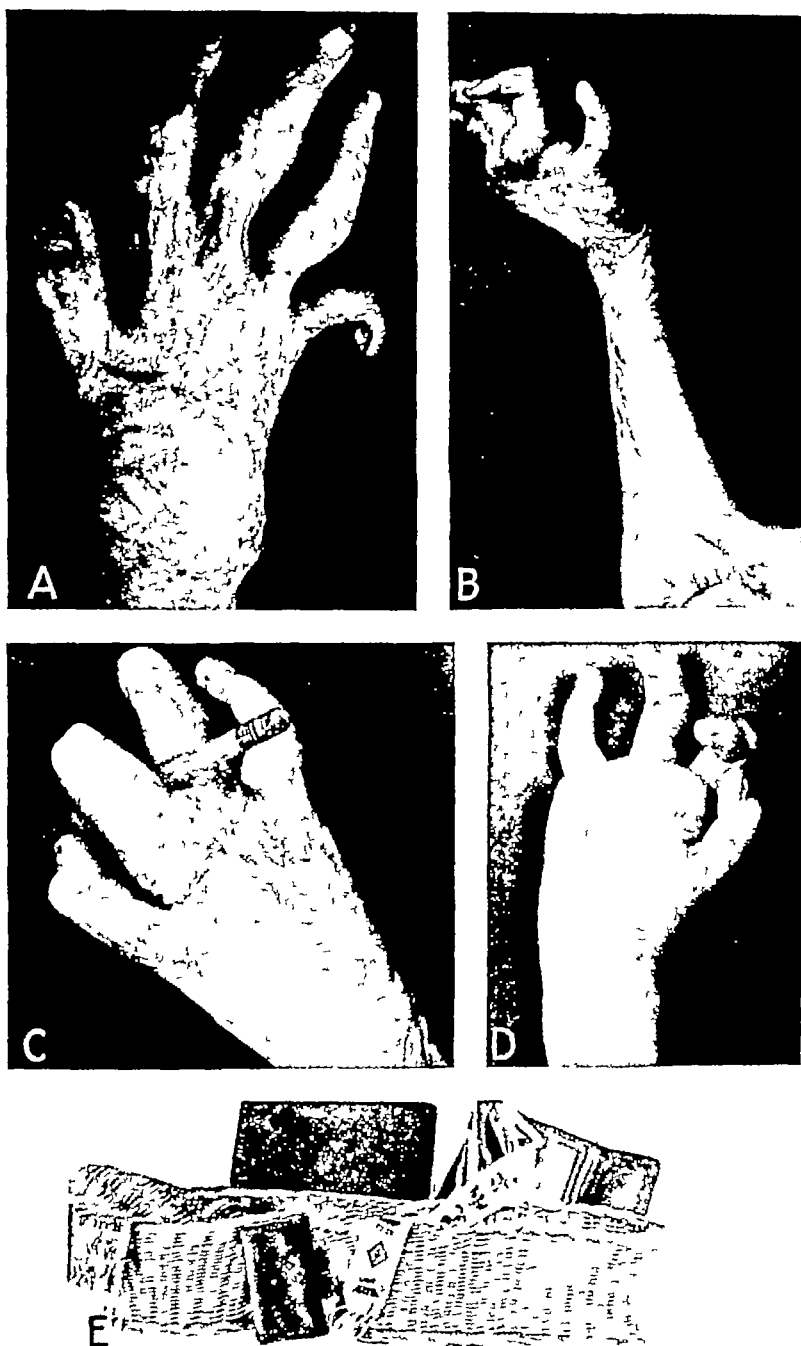


Fig 46—A and B, Complete loss of use of hand one year after severe burns which had destroyed all the skin and many of the tendons and joints Patient unable to dress himself C and D, After several operations by which the scar skin was removed and replaced with split grafts Appearance not good, but patient able to draw well enough to enter art school E, Part of many objects made as a result of instruction by occupational therapists (author's article in Ann Surg., Vol 107 J B Lippincott Co)

dislocations and deformities of joints would occur. This is the result of widespread loss of the surface, but the depth of scar in such a case cannot be determined until the dissection of it has progressed. In this

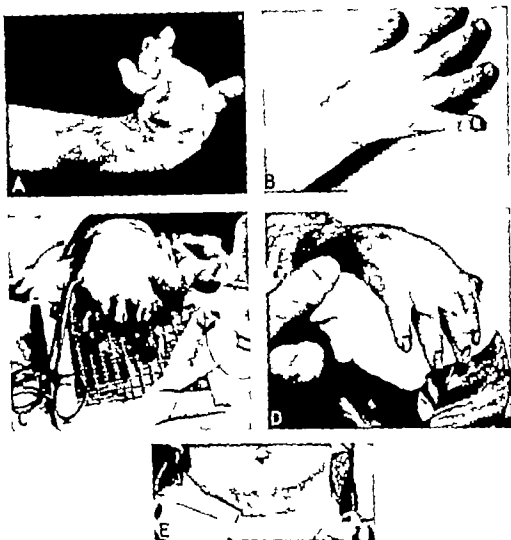


Fig. 47—A, Complete extension deformity following widespread loss of the full thickness of the skin, with thumb and fingers definitely out of position of function. B, Full restoration of function after careful dissection and coverage with a single full thickness graft. Shown after two years to indicate the permanence of the result. There is complete flexion of the wrist and of all carpal joints. C, Method of fastening hand to wire (or solid aluminum splint) with sterile adhesive tape and of application of the graft (three hours operative time). D, Three weeks after operation, full take of graft; arm being taken off splint. E, Abdominal defect healed by direct suture (author's article in *Ann. Surg.* Vol. 107 J. B. Lippincott Co.)

instance, complete function and movement were restored with a single free full thickness graft from the abdomen, put on at one operation and covering the entire area (Fig. 47 B). At operation in a situation

of this sort, the scar is very carefully dissected from the underlying tendons, rough, forceful flexion of the wrist (or of any other region that is being opened adjacent to a joint) will tear the scar that overlies the tendons and this will cause undesirable exposure of the tendons themselves. The scar is removed in layers if necessary, with crosscuts in small areas to allow stretching, and the contracted tendons are gradually pulled out so that complete flexion can be obtained if possible. It is even advisable to leave a thin layer of scar on the tendons rather than to expose them, if a free graft is to be used, because the graft will not grow on a large area of exposed tendons. When all relaxation possible has been obtained, the hand is fastened to the wire mesh splint, as illustrated (Fig. 47, C), or to an aluminum splint prepared ahead of time, using sterile adhesive tape. The splint is bent to throw the wrist and fingers as far out of the former contracted position as possible and an accurate pattern of the defect is cut. A full-thickness graft is then sutured all around the edges and up on the fingers (Fig. 47, C). The hand is represented in Fig. 47, D, after the lapse of three weeks, with full take of the graft, it is now ready to be removed from the splint and function can be started.

If the defect will not open completely at the first attempt because of tendons that are too tight, or for fear of exposing too much of them on forced manipulation, then grafts are applied to whatever areas are open and further freeing and grafting are done subsequently. If tendons are lost and have to be replaced, or if they are too much exposed, then the advisability of using a thicker, pedicled flap must be considered (Fig. 48).

Losses of the palmar surface nearly always can be repaired with free grafts. For deep losses along the fingers repair with thicker flaps may be required but full function in a case of this sort is seldom obtained. The contracted single finger, especially that of a child, if keloid has formed, with its probable accompaniment of deep inflammation, may be one of the hardest deformities to repair, and careful consideration always should be given this apparently simple lesion.

The hand in Fig. 30, A represents complete flexion deformity and webbing of the fingers. Restoration was obtained with one full-thickness graft and two subsequent operations for release of the webs (Fig. 30, B, C). The appearance at the time of the first dressing is shown in Fig. 30, D, E, F. The fingers have been held with silver transfixion wires around the terminal phalanges and fastened to the wire splint. The wire is used instead of tape when the graft comes as far out on the fingers as it did in Fig. 30. A pad of gauze separates

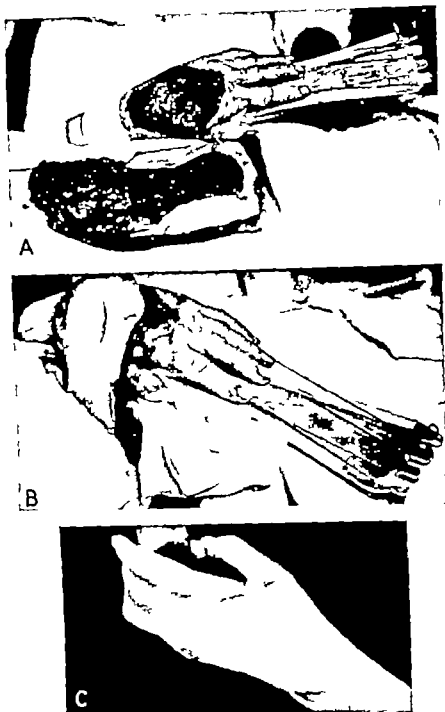


Fig. 48.—A and B, Shotgun wound of hand, with repair by a direct pedicled flap from the abdomen. Hand splinted to give traction for the fingers, and thumb rotated. Same splint used after hand is detached to obtain motion of joints and to maintain traction. A small wound of entry on the palmar surface was left to close itself. C, Result is a hand that is far superior to any prosthesis that might be devised (author's article in *Ann. Surg.*, Vol. 107 J. B. Lippincott Co.)

the dorsal surface from the wire splint, over the knuckles, a thin rubber sponge may be used.

The question of late function of a free graft in the palm might well arise and, in the answer, the relevant anatomy may be considered. The skin and subcutaneous tissues of the palm are different than they are elsewhere, for normal function, the thenar pad is essential, the thumb must be as strong as the fingers, and normal position and movements of the fingers are essential. When the palm is resurfaced, the skin necessarily must come from another part of the body; it always retains its original characteristics and does not change into the type of skin of the sound palm. If any work proves to be too strenuous, little can be done other than to have the patient wear a glove for protection. In resurfacing the palm, substitution of a pedicled flap seldom would be of benefit because the skin would differ from that of the palm just as it would if a free graft were used. The details of a full-thickness graft for the hand are further outlined in Fig. 38.

Pedicled Flaps for Repair of the Hand—There are definite indications for the use of thick, pedicled flaps in repair of the hand, such as deep burns from roentgen rays, gunshot wounds, any deep wound that exposes too much tendon, bone or joint, or any late repair in which, subsequently, work will be needed on tendon or bone, to accomplish a result which could not be attained by restoration of the surface merely with a free graft. A broad, or double, pedicle nearly always can be arranged so that the flap does not have to be delayed.

Probably as many pitfalls occur in the use of flaps as in the use of free grafts. Patients frequently are seen with inadequate flaps, which are bunched up in a pad on the hand, replacing only part of the deformity.

One of the necessary uses of a flap is illustrated in Fig. 48, A. The hand had lain directly over the muzzle of a shotgun when the gun had been discharged. A small wound of entry was present in the palm but huge excavation of the dorsum resulted. Amputation had been considered but it was suggested that at least an attempt might be made to save the hand. Accordingly, after eight days of cleaning the area, a direct flap from the right lower abdominal quadrant was sewed in place. Important points, shown in Fig. 48, A, B, are that the hand was on a splint from the start; the splint was even carried across the abdomen. The fingers were held extended by means of traction exerted by rubber bands passed through holes in the finger nails and passing to simple notches on the end of the splint. Traction was maintained by this same means during the entire period of attachment of four weeks; otherwise, the fingers would have retracted for a distance

equivalent to the length of that portion of the metacarpals which had been lost; also, marked bunching of the flap would have taken place. Another important point is that the splint was cut out to allow rotation of the thumb. This type of splint can be employed *after operation* to gain freedom of the joints, by simply bending the splint with the hand on it. The result in the case represented in Fig. 48, C was a



Fig. 49—A, Arm had grown solidly to body; excessively thick scar extended over the entire side; a deep, dirty sinus ran high into axilla. B, C, and D Repaired in two operations in which the region was opened and in two operations in which split grafts totalling 221 square inches (about 1,425 sq. cm.) were applied. Function was almost completely restored and the donor sites on the thighs healed excellently (Brown, Blair and Hamm: *Surg., Gynec. and Obst.*, Vol. 56)

hand that was useful in all work, far superior to any artificial one, and superior to any possible result that could have been obtained with a free skin graft.

Axilla, Chest, Body, Arms, Popliteal Areas, Legs

All of these areas, if there are large open surfaces within them, can be cleaned practically the same way as has been described and covered satisfactorily and permanently with thick, split grafts in rela-

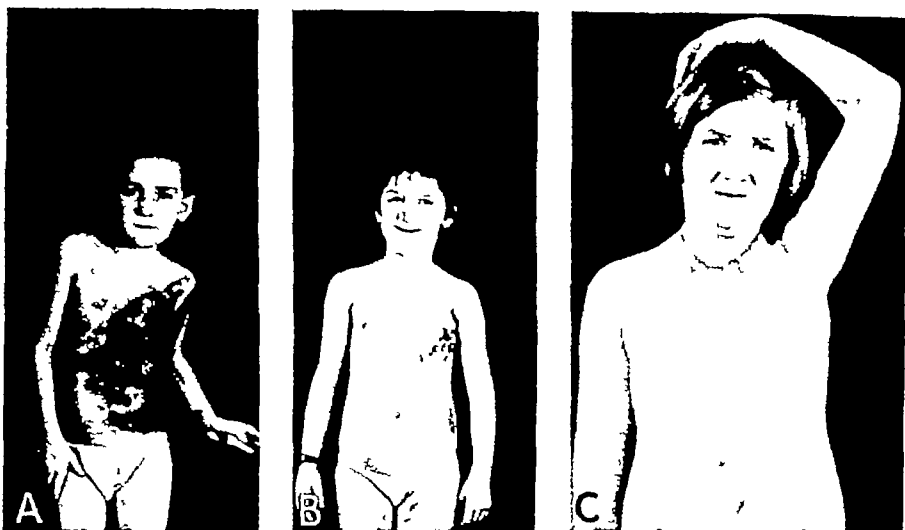


Fig 50 —A, Loss of a very large portion of the full thickness of the skin, with secondary contracture B, Complete healing in five months, after two operations in which split grafts were applied and, in the postoperative period, growth of the arm to the side was prevented C, Complete function and permanence of original repair, a year and a half later (author's article in *Internat. Abst. Surg.*, Vol. 67)



Fig 51 —Same child as the one represented in Fig 40. Generalized shortening of skin throughout the trunk and axilla, relieved by one operation on the axilla and one simple operation in which the scar on the hip was opened between the arrows. The latter operation allowed the edges of skin to retract, then the open area was immediately covered with thick split grafts as outlined. Fresh donor sites visible on thighs (author's article in *Internat. Abst. Surg.*, Vol 67).

tively few operations. It is recognized that this view is at variance with that of some authors, who have stated that full thickness grafts, or even pedicled flaps, are necessary for repair of axillary and popliteal areas. However the axilla was the region in which it was first shown that thick, split grafts were applicable for repair of large



A



B



C

Fig. 52.—A, Deformity of neck and face from scar following burn. Scar extended from arm across axilla and had pulled skin that normally is under the jaw clear down to the clavicle. B, Correction of deformity with one full-thickness graft let in low down. Surface unevenness left for later correction. C, Completed correction with full-thickness grafts done in two stages to avoid injury to lower branch of seventh nerve (Brown and Blair: *Surg., Gynec. and Obst.*, Vol. 60)

defects that overlay powerful muscles of flexion, especially if the region was to be covered by clothing. These areas are best able to overcome secondary contractures because of the forceful activity that is possible in them. Bad contractures in these areas may require such extensive dissection in freeing them, and the resultant edges and sur

face may be so rough, that immediate grafting is not practicable. The fresh wound which results can be kept clean and covered for a few days until the area has smoothed out; or the delay may be longer until a good, firm, granulating surface is established (Figs. 49, 50 and 51)

Face and Neck

Raw areas of the face usually heal quickly, because of this, they are often allowed to do so and then repairs are made later. This procedure is somewhat influenced by the involvement of the features which may require repair by pedicled flap or full-thickness graft.



Fig 53—Contracture of mouth and neck that presented a difficult situation. It was repaired with three thick split grafts from the clavicle to the level of the nose. Contour has remained satisfactory after a period of growth of thirteen years (Brown and McDowell. Surg, Gynec and Obst., Vol 72)

However, if there is continued pain or other reason for hurrying the healing, any area on the face can be cleaned and a graft used temporarily, pending final repair of any damaged features*.

It is definitely best not to use Reverdin or small, deep grafts on exposed surfaces of the face, neck, arms and hands.

Primary repairs of open areas on the neck, by split graft, are seldom satisfactory in the end because of surface roughness, contracture and unsightliness. If the area is large and requires too much time for healing, split grafts can be used first to prevent too much deformity, and final correction can be made later with full thickness grafts (Figs 39, 42, 52, 53).

* Burns of the eyelids are of especially frequent occurrence in war and they require special attention

Penis and Inguinal and Anal Regions

When the skin of the penis has been lost it is thought that free, thick, split grafts may suffice for suitable repair in most instances.



Fig. 54—Restoration after a large extremely painful loss. A single split graft was sewed on carefully. The dressing was kept wet by application of saline irrigations for four days. The final result is practically normal skin (except for hair) that can be used in the reconstruction of the ear (author's article in *Internat. Abstr. Surg.*, Vol. 67)

Such grafts could be used to effect early healing in cases in which ulceration had taken place, even if repair by a thicker pedicled flap might have to be done later.

In uneven areas that are heavily contaminated, such as these, the main preoperative preparation necessarily will be cleansing by soap and water and saline baths. The grafts will have to be applied carefully and usually will have to be held in place in the inguinal and anal regions. Large gauze folds must be anchored over them with heavy stay sutures tied from side to side. In this instance the main asepsis is firm pressure, much as it is when a stent graft is used in the mouth.

Scalp and Bare Bone

On the scalp, scar epithelium is slow to form and usually gives an unsatisfactory surface, with a marked tendency toward repeated ulceration. Probably carcinomas develop most frequently in burn scars of this region. Thick, split grafts will suffice for early and permanent covering in practically all cases. Even in the total restoration of foreheads there is little advantage in full-thickness grafts.

Free skin grafts will grow on viable periosteum, but will not grow over bare bone. Therefore, when bone is exposed, growth finally may occur by a bridging over of scar epithelium, or it may be necessary to wait for separation of the fragments of bone if there is necrosis (Fig. 54).

PERSISTENCE OF FUNCTION OF SKIN GRAFTS

Function has been found to persist in large skin grafts that have been in place during long periods of growth. Both thick split and full-thickness grafts do seem to grow, or at least to stretch out, with growth of the body area and to permit normal movement, if they have been successful from the start (Figs 55, 56 and 57).

By *satisfactory late function of the grafts is meant*: (1) Enough skin for free movement, (2) moderate looseness, (3) ability to withstand the usual trauma of moving about; (4) the development of normal sensation. Full normal sensation usually develops in free skin grafts. It is influenced by the amount of deep scar that is left and, of course, is dependent on there being sensory nerves in the area.

Metaplasia of grafts (and flaps also) does not take place and, therefore, a really normal sole of the foot, for instance, cannot be restored. This area is specialized to the point of being an organ; the skin and subcutaneous tissues are different from others from birth, and the peculiar bearing qualities are not developmental. A graft or a flap on a sole may make calluses (or even annoying warts) but it will not undergo metaplasia into true skin or subcutaneous tissue of the area. The grafts or flaps always have to be protected and forma-



Fig. 55.—A type of deformity that is most difficult to correct, with heavy distorting scar encasing entire arm and wrist. Five split grafts were required but complete function of the skin grafts persists after a period of growth of five years (Brown and McDowell: Surg., Gynec. and Obst., Vol. 72)

tion of warts guarded against. If hair is transplanted, it will continue to grow, although it may be worn off.

Skin grafts transplanted to surfaces where normally there is mucous membrane such as the mouth, larynx and eye socket, show



a



b

c

d

Fig 56—*a, b, c*, Complete circular loss repaired in three operations in which split grafts were applied. In *b* and *c* the condition after two years is represented, *d*, legs of patient with same type of burn eight years after a single operation (Brown and McDowell Surg, Gynec. and Obst., Vol 72).

no evidence of undergoing a change to mucous membrane. The skin simply persists as such, and hair even grows in these areas if there are any functioning follicles in the graft.

The results of grafting vary in different regions and with the type of lesion. A graft that is put on a soft base with a good blood supply

can be expected to be more nearly certain of taking than if it is put over the shin or ankle, where there is but little deep pad to help absorb shocks and trauma. Likewise, an old burn from roentgen rays or an ulcer of the leg, with deep and surrounding fibrosis and lack of resiliency of the tissues, does not afford as good a bed or area for a graft as a simple burn. Old chronic ulcers of the leg with deep scarring and edema tend to recur, so that improvement of local circulation is necessary if the repair is to prove efficient, the graft itself, of course, may assist in this. A frequent cause of loss of grafts that are applied for fibrosed ulcers of long standing is failure to remove the scar down to soft tissue. Denuding bone and tendons should be avoided in the process, however.



Fig. 57.—Restoration in one split graft, preceded by correction of condition of bone by Dr. Crego. Function satisfactory with graft on sole of foot after nine years of growth. Free grafts can be successful on the foot only if sufficient deep pad has been left; otherwise, a thick pedicled flap is used (Brown and McDowell Surg., Gynec. and Obst., Vol. 72)

Although free grafts and flaps are not being compared, a successful graft may be more likely to continue satisfactorily throughout a period of growth than a pedicled flap, because flaps have a tendency to hump and become heavy from fat rather than to spread out.

Cosmetic results with free skin grafts may not be as good as the functional results described. What would be a good functional result on a leg or axilla would be a very poor one on the face, because of roughness and pigmentation. However, the same general rule applies for a late, desirable cosmetic result as for function, if the graft has been satisfactory soon after operation, it will probably persist as such. The unevenness and wrinkling can be minimized by careful dissection of the base and by using full-thickness rather than split

grafts. However, chances of too much pigmentation persist without relief at the present time, and the patients may have to rely on application of cosmetics.

RELEVANT ARTICLES

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- 3 Blair, V. P., Brown, J. B., and Hamm, W. G · The Early Care of Burns and the Repair of their Defects J.A.M.A. 98 1355-1359 (Apr 16), 1932
- 4 Brown, J. B · Homografting of Skin: With Report of Success in Identical Twins Surgery 1· 558-563 (Apr), 1937.
- 5 Brown, J. B Restoration of the Entire Skin of the Penis Surg., Gynec. and Obst. 65· 362-365 (Sept.), 1937.
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CHAPTER V

BURNS IN CHEMICAL WARFARE

DAVID P. BARR, M.D.

BURNS of varying depth and extent may be produced by a wide variety of incendiary materials and by the vesicant chemicals, for example mustard (dichlorethyl sulfide) lewisite (dichlorobis(2-chlorovinyl)arsine) and ethyldichlorarsine. Burns resulting from incendiary substances such as thermit, white phosphorus, the combustible oils and the combustible metals may be deep and serious but their symptoms, prognosis and treatment do not differ significantly from those of other burns of like degree that result from heat. Exposure to the vesicant agents, on the other hand, offers some special problems.

MUSTARD

Mustard (dichlorethyl sulfide) is an oily, volatile liquid. Extensive destructive lesions of the skin may result from exposure to small amounts of the liquid or from prolonged exposure to even low concentrations of the vapor. The chemical is soluble in fats and is absorbed with great rapidity by the oils of the skin. The essential injury probably occurs as soon as mustard has penetrated the surface but the evidences of a burn may be delayed for an hour or more after contact with the liquid and for twelve hours or longer after exposure to the vapor. Although the deeper layers of the dermis are usually involved, burns from mustard seldom extend below the true skin. The first symptom may be severe itching of the skin, soon followed by an erythema on which small and large blisters develop. In lesions from liquid mustard, the blisters may be arranged in a ring about a central, blanched, indurated area. Burns require some weeks to heal and secondary infections are of frequent occurrence.

Immediate Treatment

Because of the rapidity with which mustard is absorbed, abortive measures must be instituted immediately after exposure. 1. Clothes should be removed at once to prevent continued contamina

tion. Attendants should be protected by masks, gas proof gloves and protective clothing or protective ointment. The clothes should be placed in a covered metal container until they can be decontaminated.

2. Great care must be taken not to spread any of the liquid mustard which may remain on the surface of the skin. Dry pads or dry cotton swabs should be gently applied to all exposed areas. Burns are particularly likely to develop in parts where the skin is oily, moist, thin or subject to friction and these parts should receive special attention.

3. As soon as the surfaces have been dried, the skin should be dabbed gently and repeatedly with sponges moistened, but not dripping, with gasoline, kerosene, carbon tetrachloride or alcohol. It must be remembered that these are only solvents and not neutralizing agents and that therefore fresh sponges must be employed for each application. All materials used in drying and sponging should be burned or buried.

4. Substances containing chlorine cause mustard to decompose. Bleaching powder (1 part to 2 parts of water), commercial bleaching solutions or Dakin's solution should be applied directly to contaminated areas. These chemicals should not be left on for more than a few minutes because of their irritating qualities. They are of use only to prevent continued exposure and their use is contraindicated if erythema already has developed.

5. Following the treatment with solvents and chlorinating agents, the skin on and around the exposed areas should be washed thoroughly with soap and water and then gently dried.

If erythema already has developed before treatment can be instituted, solvents (gasoline, kerosene, carbon tetrachloride, alcohol) still should be applied by the same careful technic which has been described, and their application should be followed by gentle washing with soap and water. Chlorinating agents will only aggravate the irritation and must *not* be used on the erythematous skin. Itching, which is often intense at this stage, can be alleviated by hot applications or by the use of antipruritic ointments such as:

	gm or cc
Butyn sulfate	0.5
Benzyl alcohol	12.0
Menthol	0.2
Oil of lavender	0.2
Alcohol	5.0
Stearic acid	12.0

Later Treatment

All blisters on the skin should be opened, the fluid and dead skin removed and the injured area blotted with sterile gauze. On all parts

of the body except the hands, face, flexor surfaces, perineum and genitalia, the denuded surfaces should be treated with agents and by techniques recommended for other types of burns. It is recommended that 10 per cent tannic acid be first applied, this to be followed immediately by application of a mixture of equal parts of 10 per cent tannic acid and 10 per cent silver nitrate. The treatment with the mixture should be repeated four times at intervals of a half hour.

If this procedure cannot be carried out, a 2 to 5 per cent solution of tannic acid can be applied by sponging or spraying. Tanning also can be accomplished by a solution of compound tannic acid powder which has the advantage of keeping well, either in the dry state or in its balanced saline solution. The powder and its solution can be prepared as follows:

<i>Compound tannic acid powder</i>	gm. or cc.
Potassium chloride	0.42
Calcium chloride	0.84
Salicylic acid	1.0
Sodium chloride	10.5
Tannic acid	100.0

Finely powdered materials should be ground thoroughly and mixed intimately in a mortar; then they should be placed in a bottle and shaken thoroughly to complete the mixing.

To make a compound tannic acid solution all of the powder should be dissolved in 1000 cc. of distilled water. The solution will be rendered antiseptic and stable by the salicylic acid and can be kept in this form.

On the face, hands, flexor surfaces, perineum and genitalia, aniline dye should be applied according to the technic developed by Aldrich. A mixture is made of equal parts of the following dyes:

Brilliant green	1:400
Gentian violet	1:400
Neutral acriflavine	1:1000

This mixture can be sprayed on the surfaces at intervals until a satisfactory eschar has formed.

An optional treatment of burns of the hands, face and genitalia is application of 5 per cent sulfadiazine in an aqueous suspension or in boric acid ointment.

If infection occurs under the eschars, the tanned surface should be removed and the area dressed with an 0.08 per cent solution of sulfanilamide in physiologic solution of sodium chloride.

LEWISITE

In its physical characteristics, lewisite (dichlorobis(2-chlorovinyl)arsine) is similar to mustard but is more volatile. It is more immediately irritating. Symptoms develop earlier and are more severe. After contamination with the liquid, redness of the skin appears within one to fifteen minutes. Blisters form early and may reach their maximum within twelve hours. Until neutralized or removed, lewisite continues to penetrate, extending through the skin and subcutaneous tissues to the muscles and other structures. In addition to the painful burns, which frequently become infected, symptoms of arsenic poisoning may be expected. These include dryness of the throat, diarrhea, restlessness and, in some cases, paralysis.

Immediate Treatment

To be effective, treatment must be instituted at the earliest possible moment and should be begun while the clothes are being removed.

The exposed areas should be swabbed repeatedly with hydrogen peroxide. This should be used in 8 per cent solution.* Fresh swabs should be used for each application. Later these should be destroyed. If necessary, water can be substituted for the alcohol.

When the swabbing has been completed, the areas should be thoroughly washed with soap and water.

For the intense itching which accompanies the early stages of the burn, the antipruritic ointment described for the treatment of burns from mustard may be helpful.

When burns produced by liquid lewisite are seen soon after they have become erythematous, multiple scarification of the contaminated areas, together with immediate suction, should be instituted. If the area of contamination is small and strictly limited to an easily mobilized portion of the skin, wide excision down to the subcutaneous tissue is indicated.

Later Treatment

Blisters can be treated by draining and removal of the cover. If the patient survives the general intoxication of lewisite, the later treatment does not differ from that of deep burns due to other causes.

ETHYLDICHLORARSINE

Vesiculation of the skin with ethyldichlorarsine is less marked than with lewisite or mustard. Therapeutic measures must be insti-

* 30 per cent superol, Merck, diluted to 8 per cent with water.

tuted promptly. They do not differ from those recommended for the early treatment of burns from lewisite. Arsenical poisoning may follow exposure to the liquid.

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SHOCK

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PREFACE

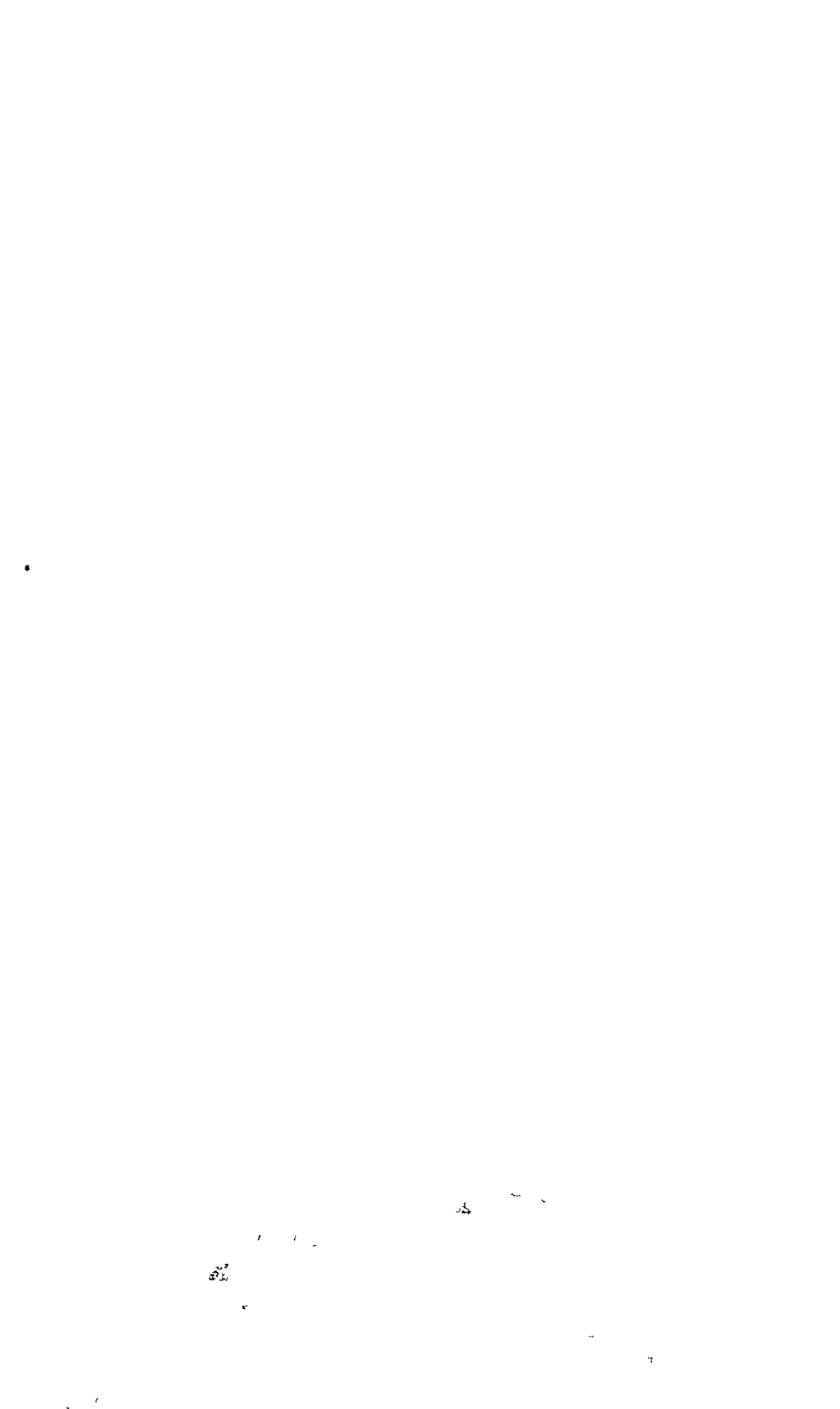
ALTHOUGH the incidence of shock in civil practice has been greatly reduced in recent years, this condition is still one of the major problems in military surgery. With the increasing use of mechanized equipment and high explosives, the incidence of shell wounds and crushing injuries which produce shock is naturally greater. In addition, modern warfare is admittedly one of rapid movement so that the facilities for the early treatment of the wounded afforded by well-equipped hospitals may be lacking. It is well known that the process of shock brings about certain changes in the function of the tissues which, after a time, become irreversible. It is accordingly essential to recognize shock in its incipient stages in order to prevent its development and to start treatment at the earliest possible moment. Since large, stationary medical units will not be available, the early treatment of shock must be carried on by small, mobile units. This manual on shock has been prepared for the use of the Army and Navy with the realization that the prevention and treatment of shock must perforce be carried on by the medical personnel who are in immediate contact with the fighting forces.

The subject of shock will be considered in three sections: (1) its nature and mechanism, (2) its prevention and treatment, and (3) the use of intravenous fluid therapy. Emphasis will be placed on practical considerations. In discussing the mechanism of shock, no effort will be made to review all the controversial details involved in the various theories which have been advanced to explain this condition. This limitation has been imposed in order to relate the consideration of the mechanism more closely to the prevention and treatment of shock. For the purpose of simplicity references will be omitted.

SUBCOMMITTEE ON SHOCK
Committee on Surgery
Division of Medical Sciences
National Research Council

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SHOCK

CHAPTER I

THE MECHANISM OF SHOCK*

NORMAN E. FREEMAN M.D

DESCRIPTION AND DEFINITION OF SHOCK

Clinical Features of Shock

The patient in shock has the appearance of being seriously ill. The significant features are centered about the peripheral circulation. The skin is cold, moist, and of an ashen gray or cyanotic hue. The pulse is feeble and rapid. There is usually a lowered blood pressure although, in the early stages, the pressure may not be significantly reduced. The patient presents the picture of weakness bordering on exhaustion. This condition is not sudden in onset but takes time for its development. During the downward course, there is progressive enfeeblement and gradual suppression of all the bodily functions.

Physiologic Features of Shock

Peripheral circulatory failure is the characteristic physiologic feature of shock. This failure of the circulation is caused by a discrepancy between the capacity of the vascular system and the volume of fluid which it contains. The blood is composed of red blood cells suspended in plasma. The blood vessels under normal conditions are relatively impermeable to the proteins of the plasma and it is the colloid osmotic pressure of the plasma proteins which attracts fluid from the tissue spaces and holds the fluid portion of the blood within the vessels. Any condition which brings about the loss of blood or plasma, such as occurs after hemorrhage or after severe burns, facilitates the development of shock. In addition, any agency which affects the permeability of the blood vessels and allows the escape of plasma protein from the blood stream will lead to the development of shock.

Definition of Shock

Shock may, therefore, be defined as the clinical condition characterized by progressive reduction in circulating blood volume due to increased capillary permeability

* From the Harrison Department of Surgical Research, Schools of Medicine, University of Pennsylvania.

HISTORICAL SURVEY

Two broad concepts have been advanced to explain the mechanism of the increased capillary permeability: traumatic toxemia and reduced circulation.

Traumatic Toxemia

The theory of "traumatic toxemia" as the cause of shock was advanced on clinical grounds by Quénu and on experimental evidence by Cannon and Bayliss in 1917-1918. It was based on the investigations of Dale, Laidlaw, and Richards on shock produced by the injection of histamine. According to this concept, a toxin was absorbed from traumatized tissue and was carried by the circulation throughout the body. This toxin caused injury to the capillaries. The initial loss of circulating blood volume in traumatic shock was ascribed to the increase in capillary permeability which resulted from this injury.

Reduced Circulation

The theory that "reduced circulation" was the cause of shock was advanced by Erlanger, Gesell, Gasser, and Elliott, and was based on their observations that shock was always associated with a reduction in circulation. It was their belief that the progressive reduction in circulating blood volume was produced by segregation and stagnation of blood.

The reduced circulation found in shock could be explained either by the loss of blood or plasma into the area of injury or by reflex vasoconstriction.

Local Loss of Fluid—Blalock and many others have demonstrated the significance of local loss of fluid into the region of injury in amounts sufficient to account for shock from reduced circulation.

Vasoconstriction.—Freeman and his associates found that reduced circulation was consistently associated with shock in clinical cases. They have presented evidence that vasoconstriction, such as is produced by the injection of massive doses of epinephrine, could result in a reduction of blood volume.

Initiating versus Sustaining Factors in Shock

It is generally agreed by all those who are working in the field of shock that loss of blood, plasma, or body fluid is the *most significant initiating factor* in the production of shock. Reduced circulation, caused by this primary loss, brings about various physiologic and chemical changes which may be regarded as the *sustaining factors* in shock.

Time Factor in Development of Shock

Reduced circulation leads to dilatation of the capillaries with an increase in their permeability so that plasma escapes from the vessels. The blood corpuscles are thus segregated and "lost" from the circulation. To this change in capillary permeability from anoxia may be ascribed the progressive reduction in circulating blood volume which constitutes the process of shock. Time is required, however, for anoxia to have its effects on capillary permeability and this time factor is of the utmost importance both in the mechanism of shock and in its treatment. It is well recognized that the treatment of simple dehydration or even of hemorrhage by replacement of the lost fluid brings about recovery. If the process has continued for a period of time, however, so that changes in capillary permeability have been produced, recovery cannot be readily attained by simple replacement. The importance of *prevention of shock by early treatment* can hardly be overemphasized.

CRITERIA OF SHOCK

It is essential to establish certain criteria of shock to furnish a guide for treatment of the clinical condition.

Blood Pressure

This is the first criterion which will be discussed since blood pressure can readily be determined both in clinical cases and in animal experiments. Cannon has stated "one of the central problems, if not the most important central problem, of shock is that of discovering the reason for the lowered arterial pressure." In a recent monograph Blalock has written that in shock "the blood pressure undergoes characteristic alterations, the most constant of which is the reduction in pulse pressure. In the early stages, there is a slight to moderate decline in the systolic level often accompanied by a slight rise in the diastolic value. As the condition progresses, both pressures decline—the systolic more than the diastolic. In the advanced stages the decline becomes so pronounced that the blood pressure cannot be measured, the pulse being imperceptible and the Korotkoff sounds inaudible." The question may be asked, "Why is not the blood pressure alone the only criterion of shock that is needed?" The reason that blood pressure alone is not entirely a satisfactory criterion of shock is that a decline in blood pressure may not occur until late in the course of shock, when the condition is well established and when the most opportune time for treatment has already passed. It has been shown that the cardiac output, and thus the circulation to the

body as a whole, declines after hemorrhage, burns, or trauma well before there is a significant fall in blood pressure. It is probably true, on the other hand, that a sustained blood pressure in clinical cases below 80 mm of mercury, the critical level emphasized by Cannon, "is no longer capable of maintaining an adequate volume-flow to the tissues, and thus serving the normal oxidations of the body." In experimental studies, as Blalock has pointed out, "if one is determining the quantity of the local loss of blood and fluid that results in fully developed shock, a sustained reduction in the blood pressure to or below the critical level is a good criterion."

Blood Flow

Probably blood flow is a more accurate criterion of shock than is blood pressure. As mentioned above, the cardiac output has been found to decline well before the blood pressure falls in shock produced by hemorrhage, burns, or trauma. The fall in blood pressure is prevented by vasoconstriction, which acts as a compensatory factor. The constriction of the arterioles, however, causes a reduction in the peripheral blood flow and thus facilitates the development of tissue anoxia. The time relationship between the decrease in peripheral blood flow and the fall in blood pressure observed in a series of experiments is illustrated in Fig. 1. The extremities of the dogs, anesthetized with ether, were traumatized but excessive loss of blood and plasma into the region of injury was prevented by bandaging and applying adhesive tape to the extremities to be traumatized. As can be seen, the first reaction which was displayed by the dogs after the trauma was a marked reduction in blood flow, as measured by the plethysmograph, through the uninjured hind paw. This reduction in circulation took place well before there was a serious fall in blood pressure. There was a close correlation between the state of the peripheral circulation and the incidence of shock. In fourteen dogs which went into shock the blood flow was severely depressed. In the two dogs which failed to go into shock a comparable reduction in peripheral blood flow was not observed. The reduced oxygen saturation of venous blood obtained from the right heart indicated the diffuse character of the tissue anoxia caused by the reduced circulation.

Clinical Estimations of Reduced Circulation.—Objective methods of estimating reduced circulation are time-consuming and laborious. Determinations of cardiac output, either by the ballistocardiograph or by the Fick principle, and the measurement of peripheral blood flow by means of the plethysmograph or one of the thermo-electric

stromuhr methods are not practicable. Reliance must then be placed on clinical signs of reduced circulation as criteria of shock.

The character of the pulse is of major significance. Its volume probably gives the single best indication of the general state of the circulation. The contrast between a full radial artery which indicates an adequate output of the heart and a weak, "thready" pulse of poor

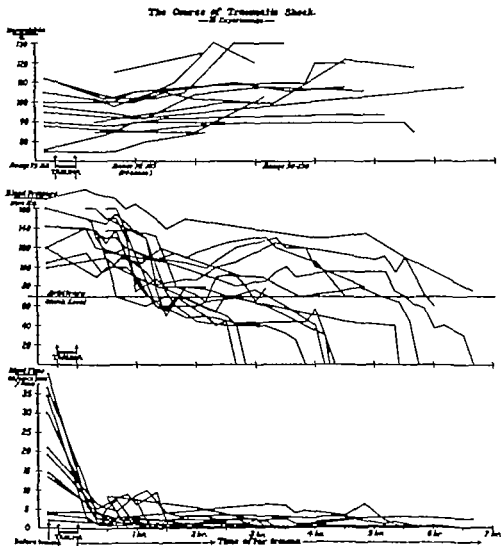


Fig. 1—The course of traumatic shock.

volume differentiates a blood pressure associated with a normal cardiac output from an identical blood pressure maintained by peripheral vasoconstriction in the face of a diminished circulating blood volume.

The surface temperature of the extremities also furnishes an index of the efficacy of the peripheral circulation. The extremities of a

shocked individual are always cold even though the internal body temperature may be elevated. In experiments on dogs in which the peripheral blood flow was measured during the process of traumatic shock, it was noted that the paws became cold approximately at the time that the peripheral circulation was reduced to the shock level.

The color of the skin and mucous membranes again furnishes good evidence of the state of the circulation. Instead of the normal healthy color spoken of as the "pink of condition," the skin is dusky, slate-colored, or ashen. At this point, however, caution should be exercised not to interpret the cyanosis associated with the administration of one of the sulfonamides for that resulting from inadequate circulation. The color of the skin of the face, especially about the mouth, is particularly significant. Vasoconstriction may readily be seen in the circumoral region before it is evidenced elsewhere. Cyanosis is generally present in the nail beds, and the skin of dependent portions of the body and abdominal wall may exhibit a blotchy appearance.

Collapsed veins have long been recognized as a constant feature of shock. The speed with which the veins fill after the blood has been stroked out of them furnishes a rough estimate of the peripheral circulation.

Bleeding from incisions or lacerations furnishes an excellent index of the state of the circulation. When the finger tip is pricked to obtain blood for study, there slowly exudes a dark, viscous fluid. Superficial wounds do not bleed, except for a slow trickle from the veins. The blood is dark. The muscles appear brown and desiccated. As Harkins has remarked, "If one were to select a single determination to follow the course of shock, an observation of the peripheral blood flow might be the best to choose."

Clinical Signs Associated with Reduced Circulation.—*Sweating* is a frequent accompaniment of reduced circulation. It is associated with stimulation of the sympathetic nervous system. This stimulation may result from reduced blood volume or it may represent one of the physiologic reactions to traumatic stimuli. In any case, the peripheral circulation is concomitantly reduced either by low blood pressure or by vasoconstriction. Distinction should be made, however, between the sweating which is associated with a full pulse and warm skin and the "cold sweat" of undue stress. The former represents one of the normal physiologic responses of the body in an effort to lose heat while the latter is decidedly harmful since it facilitates loss of body fluid at the very time that conservation is needed.

Thirst is a constant feature of shock and may represent the

physiologic response of the organism to the reduced secretions of salivary glands and the glands in the oropharynx resulting from restricted circulation. The tongue is dry and fissured. Mouth breathing frequently produces excessive drying of the mucous membranes of the mouth and tongue. In order to differentiate the reduced salivary flow associated with shock from simple surface drying the under surface of the tongue and the mucous membranes of the gums protected by the tongue should be examined.

Urine secretion is always diminished as the process of shock advances. This restriction may result from dehydration, from lowered blood pressure, or from reflex vasoconstriction. The urine, in spite of being scanty, is not as concentrated in shock as would be expected from its small volume. A good output of urine is a favorable sign. As Peters has commented, "The patient who is excreting 1000 to 1500 cc. of urine daily is seldom a subject for anxiety."

The *mental state* of the shocked patient is usually apathetic. Although consciousness is usually maintained until the end, reaction to painful stimuli, both in clinical and experimental shock, is lessened.

Blood Volume

This is even more significant than blood flow as a criterion of shock. Robertson and Bock, and Keith, measured the blood volume of wounded soldiers during the war of 1914-1918 and found it to be constantly reduced in soldiers suffering from shock. Further, the diminished blood volume was recognized as bearing a definite relation to the severity of the patient's clinical state. It is generally agreed today that there is a reduction of the circulating blood volume in shock. Experimental observations have fully confirmed the findings in patients. Reductions of blood volume sufficient to produce shock have been demonstrated after trauma, burns, intestinal obstruction, and strangulation. It must be admitted, however, that no entirely satisfactory method for the determination of blood volume in shock has yet been found. The dye method which has been employed to measure the plasma volume depends on the determination of the concentration of the dye in the plasma after the injection of a known quantity. Accuracy of results by this method depends on the assumption that there is an equal distribution of the dye throughout the blood stream and that only a small, predictable fraction of the dye escapes from the circulation during the time allowed for complete mixing. The fact that dye appears in the lymph soon after injection renders this assumption unjustifiable. Although reproducible results can be obtained with the dye method under basal conditions, the

shocked individual are always cold even though the internal body temperature may be elevated. In experiments on dogs in which the peripheral blood flow was measured during the process of traumatic shock, it was noted that the paws became cold approximately at the time that the peripheral circulation was reduced to the shock level.

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reliability of the method when applied to the study of shock is subject to grave doubt, since the increase in capillary permeability which is a feature of shock alters the rate of loss of dye from the circulation. In three experiments on traumatic shock, when this method was employed, the concentration of the injected dye, after allowing thirty minutes for complete mixing, suggested that there was even an increase in plasma volume although, at the time, the dogs were dying from shock and there was a considerable amount of blood and plasma lost into the region of injury. Computing total blood volume on the basis of plasma volume and hematocrit determination is also subject to error since an assumption has to be made that the concentration of corpuscles is the same throughout the circulation. It has been demonstrated that the concentration of red blood cells is lower in blood contained within the smaller blood vessels than in blood which is flowing from the vessels. In general, it may be said that determinations of plasma volume are fairly reliable in the early stages of shock. When they are attempted at a time when reduced circulation requires a prolonged time for mixing, and when there is wide spread increased capillary permeability, results are not dependable. Determinations of blood volume by the carbon monoxide method have been more reliable in our hands than by the plasma dye method. The concentration of carbon monoxide in the blood is measured after allowing the animal to breathe a known quantity of the gas for a period of twenty to thirty minutes. Since the carbon monoxide is combined with the hemoglobin, it cannot escape from the circulation except with red blood cells. Of course this method, too, is not dependable in case that red blood cells are being lost from the circulation, either by bleeding or by segregation of blood, during the period allowed for mixing. There is again the objection that estimation of total blood volume depends on the questionable assumption that the proportion of red blood cells is the same throughout the circulation.

Although it is agreed that methods of determining blood volume have their limitations, the significance of reduced blood volume as a criterion of shock cannot be disregarded. To emphasize this point further, some experiments will be cited. The effect of total sympathectomy on the shock resulting from trauma with restricted local loss of fluid was studied. As mentioned previously (Fig. 1), close correlation had been observed in experiments on the normal dog, between the incidence of shock and reduction in peripheral blood flow. The power of reflex vasoconstriction, however, is lost in the totally sympathectomized animal. In the first two experiments which were performed on sympathectomized dogs, the peripheral blood flow was

well maintained. The blood pressure, after a preliminary fall, was fairly well maintained for six hours. According to the criteria of blood pressure and blood flow, shock had not been produced. We* were not satisfied however with such conclusions, since examination of the tissues after the dog had been killed disclosed certain changes in the viscera typical of shock. When determinations of blood volume were made with the carbon monoxide method on these sympathectomized dogs, it was found that, even without reduction in blood pressure or circulation, a loss of blood volume had occurred greater than could be accounted for by the measured amount of fluid lost into the region of injury. In other words, neither blood pressure nor blood flow served under these circumstances as criteria of shock.

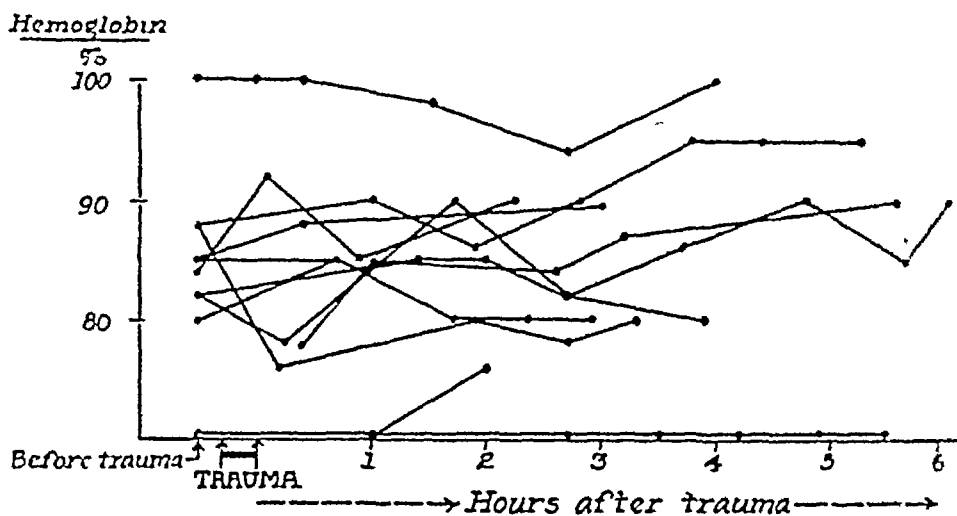
Clinical Estimation of Blood Volume.—Such estimation is not readily accomplished because of technical difficulties. Clinical signs of reduced blood volume are mainly to be found in alterations of blood pressure and blood flow. These have already been described.

Hemoconcentration.—This has been suggested as "the earliest detectable manifestation of shock as well as the most accurate index of its severity." It is probably true that concentration of the formed elements is an accurate index of shock when the initiating cause is loss of fluid or plasma from the blood stream. Examples of this condition are furnished by shock associated with dehydration, burns, or the implantation of skeletal muscle or liver in the peritoneal cavity. When, however, there has been a loss of red blood cells from the circulation combined with the loss of plasma, naturally the concentration of the formed elements in the remaining blood cannot be expected to furnish a reliable estimate of the blood volume. An example of the significance of loss of blood as a determining factor in hemoconcentration is furnished by the experiments illustrated in Fig. 2. Traumatic shock was produced in dogs anesthetized with ether as described before. In one group, loss of blood and plasma was not restricted. In the second group, local loss was reduced by compression of the injured extremities. Hemoconcentration developed only in the cases in which loss of blood was restricted. In clinical cases of shock from injuries, loss of blood is of variable degree. Hemoconcentration by itself, therefore, cannot be used as a criterion of reduced blood volume. On the other hand, it is of great significance as a criterion of increased capillary permeability when used to follow the course of shock or its response to treatment.

* See Cullen, M. L., Schecter, A. E., Freeman, N. E., and Laws, M. K., in *Blood Substitutes and Blood Transfusion*, Mudd, Thalheimer and Associates, C. C. Thomas, Springfield, Illinois, 1942.

Hemoglobin in Traumatic Shock
Unrestricted Local Fluid Loss

— 12 Experiments —



Hemoglobin in Traumatic Shock
Restricted Local Fluid Loss

— 15 Experiments —

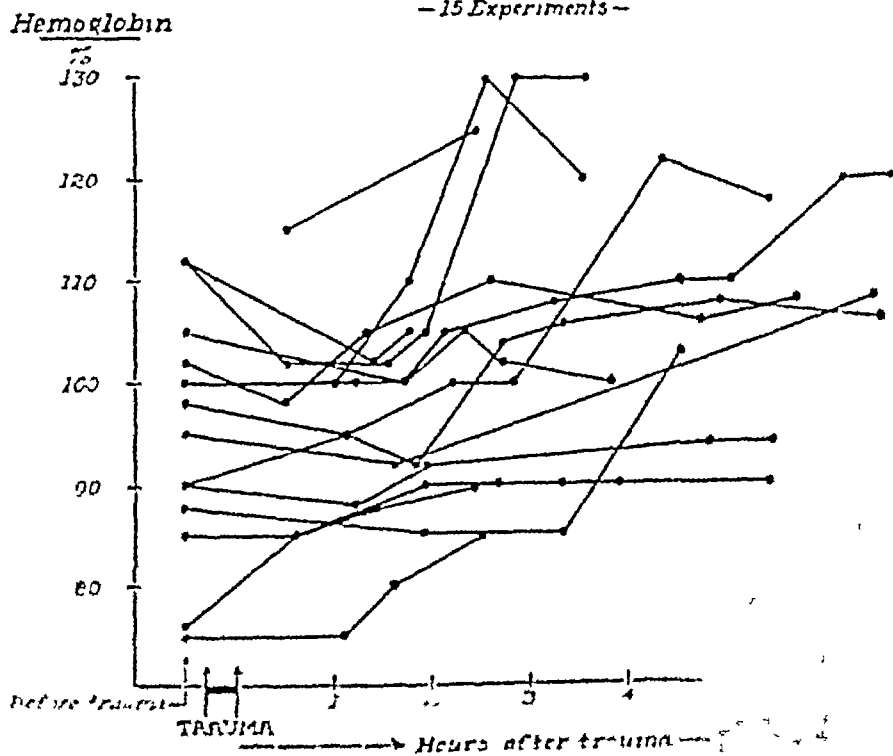


Fig 2—The effect on hemoconcentration of restricting 1 region of trauma

Increased Capillary Permeability

Increased capillary permeability as the ultimate cause of the progressive loss of volume of circulating blood, is probably the most accurate criterion of shock but, at the same time, the factor which is understood the least. Under experimental conditions it can be observed by direct visualization of the capillaries under the microscope. As the plasma leaves the vessels, the formed elements, either red blood cells or particles which have previously been injected, become concentrated as they pass to the venous side of the capillary bed. Obstruction of the venule and capillary is thus produced.

A second method of estimating capillary permeability is afforded by observing the behavior of dyes injected into the blood stream. Certain of these dyes (for example T 1824) leave the circulation only slowly. They can be seen to fill the true capillaries. With an increase in capillary permeability their escape from the blood vessels can be readily visualized.

Clinical Estimation of Increased Capillary Permeability—Increased capillary permeability cannot be determined by either of these methods of direct visualization of the capillaries. Reliance must be placed on evidences of increased capillary permeability furnished (1) by examination of the blood remaining in the vessels, (2) by the clinical signs produced by the fluid which has left the blood vessels, or (3) by the response to treatment.

Hemoconcentration is a good index of increased capillary permeability if there has not been simultaneous loss of whole blood. When the measurement of the concentration of the red blood cells, either by hematocrit determination or estimation of hemoglobin, is combined with the determination of the concentration of plasma proteins, the state of the capillary permeability can be estimated. For example, in the initial dehydration resulting from vomiting or diarrhea, fluid without plasma protein is lost from the body. Simultaneous concentration of both the red blood cells and the plasma proteins in equal degree should occur. Concentration of the formed elements more than that of the plasma proteins indicates that there has been an increase in capillary permeability. By comparison, if the initial dehydration has resulted from the loss of plasma from an extensive burn, the concentration of red blood cells would be expected to increase whereas the level of the plasma proteins would be expected not to change. Under such circumstances, an increase in capillary permeability would be demonstrated. Alterations in the specific gravity of the serum have been useful in following dilution or

tion of the plasma proteins.

Clinical signs of fluid which has left the blood vessels in the course of shock are difficult to elicit except when the fluid has been lost through the pulmonary capillaries. It then makes itself evident in the form of pulmonary edema. This condition is frequently encountered in cases of shock associated with head injury and is occasionally observed in the course of other types of shock. Pulmonary edema, however, generally is produced during the treatment of shock, when replacement of plasma is attempted by the rapid intravenous injection of solutions containing only crystalloids. Under such circumstances, the pulmonary edema can hardly be interpreted as evidence of an increase in capillary permeability. Fluid lost from the circulation during the process of shock goes chiefly into the tissues and lumen of the gastro-intestinal tract. The grayish brown, viscous fluid obtained by aspiration of the stomach, and that contained in the mucoid or even bloody stools, probably represents the fluid lost into this region. Attention has been called to "hemorrhagic enterocolitis" as a sequel of post-operative shock.

Response to treatment probably gives the best indication as to the state of the capillary permeability. This response can be gauged by (1) the simultaneous measurement of the concentration of plasma proteins and red blood cells after the injection of plasma, or (2) the clinical improvement manifested by the patient during treatment.

Intravenous injection of plasma should result in dilution of the red blood cells, with either an increase or little change in the *concentration of plasma protein*. When, however, the concentration of the formed elements is not reduced to the degree that would be expected from the amount of injected plasma, an increase in capillary permeability, allowing the injected protein to escape from the blood stream, must be assumed. By this technic it has recently been demonstrated that the increased capillary permeability associated with extensive burns may persist for twenty-four to forty hours. The possibility of washing stagnant red blood cells back into the circulation by the intravenous injection of plasma should be kept in mind.

The rapidity with which *clinical improvement* occurs when dehydration or hemorrhage is treated by replacement of the fluid lost indicates that serious changes in capillary permeability have not taken place. If the condition of reduced blood volume has persisted for a sufficient period, however, recovery through replacement of the fluid lost is not readily effected. This condition of "negative reaction to blood transfusion" is probably associated with an increase in capillary permeability.

PATHOLOGY OF SHOCK

Shock is the final phase of many clinical conditions characterized by prolonged impairment of the circulation. The pathologic picture of shock is usually complicated by that of the initial lesion which produced the process. In this discussion emphasis will be placed on only those features which belong essentially to shock.

In his investigations on the mechanism of hemorrhagic infarction, Welch made the significant observation that reduction in the arterial pressure in the mesentery of the dog produced stasis, with concentration of the blood corpuscles in the capillaries and veins of the loop of intestine supplied by the artery which was compressed. He could observe no change in the appearance of the vessel wall but found that the forward movement of the blood was checked and that stasis soon took place. Landis, in his observations on the mesentery of the frog, analyzed this reaction more closely and found that, although no changes might be visible in the blood vessel walls, there was an increase in the permeability of the endothelium so that plasma escaped and left the cells stranded. These facts have been confirmed by a number of observers and appear to be well substantiated. It is the opinion of the majority of investigators who have worked in the field of shock that this increase in the permeability of the minute blood vessels, in consequence of impaired circulation, is the central feature in the process of shock. In addition, the observation that a decrease in blood volume, not accounted for by the local loss of fluid, occurs in spite of a well maintained circulation in the sympathectomized dog, indicates that some other factor may contribute initially to the capillary stasis. The pathologic picture which is found when the tissues are examined after death from shock is that to be expected from peripheral circulatory failure. There is widespread congestion and engorgement of the capillaries and venules throughout the body.

Such a picture was found by Gasser, Erlanger and Meek in shock experimentally produced in a variety of ways. They observed in the intestinal mucosa that the "capillaries and small veins are greatly dilated and tightly packed with red blood cells." More recent studies on the pathology of shock by Moon have corroborated their observations. This congestion is found throughout the viscera and in the lungs. There is edema in the tissue spaces and effusion in the serous cavities. If the impaired circulation has persisted in the experimental animal for sufficient time there may be actual necrosis of the intestinal mucosa, and a condition described as "hemorrhagic enterocolitis" may be observed. The impairment of circulation affects other organs

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as well. Necrosis of liver cells, liquefaction of the suprarenal medulla, and congestion of the pancreas are observed. The kidneys show evidence of parenchymatous degeneration. Patches of capillary hemorrhage occur in the medulla, and numerous red cells are found within the tubules in such regions. The pathologic picture thus confirms the clinical and experimental evidence on the significance of increased capillary permeability as the essential feature of shock.

FACTORS IN PRODUCTION OF SHOCK

The factors which are significant in the production of shock will be considered in the light of the criteria of shock which have been described previously.

Factors Which Lead to Reduced Circulation

Loss of Volume of Circulating Blood HEMORRHAGE.—The fact that hemorrhage is of great importance in the production of shock has long been appreciated. Not only is there an immediate loss of volume of circulating blood, but the fall in blood pressure with compensatory vasoconstriction leads to a serious reduction in circulation. If the circulation remains deficient for a sufficient length of time, changes are produced, probably secondary to the tissue anoxia, leading to an increase in capillary permeability. As a result of this alteration in the capillaries, the classical picture of shock can be produced. Efforts recently have been made to differentiate shock from hemorrhage, particularly on the basis of the postmortem findings. While it is true that the physiologic and pathologic picture after acute hemorrhage is different from that seen in shock, it has been shown that if time is allowed for the development of the process of tissue anoxia by means of slow and moderate hemorrhage, the characteristic picture of shock can be produced. Blalock demonstrated that the gradual withdrawal of blood from the dog under local anesthesia in amounts sufficient to maintain the blood pressure at the critical level for a period of hours will produce shock. Freeman and his colleagues have confirmed the work of Blalock and have related the process to the reduction of circulation produced by the hemorrhage. Shock, leading to death, with characteristic hemoconcentration and pathologic changes in the tissues can be produced by slow bleeding, with subsequent reinjection of the blood. It does not seem to matter whether the hemorrhage is outside of the body, into one of the body cavities, or into the tissue spaces, as far as the production of shock is concerned.

LOSS OF PLASMA—Loss of plasma is probably even more sig-

nificant as a factor in the production of shock than is the loss of whole blood. Not only is there a reduction in the volume of circulating blood but, in addition, there is an increase in the viscosity of the blood which jeopardizes the nutrient flow to the tissues. Loss of plasma is produced by any factor which increases the capillary permeability. Direct injury of the capillaries may be produced by thermal agents, direct mechanical trauma, chemical and bacterial irritants, and tissue anoxia. The concentration of protein in the fluid which escapes from the injured capillaries has been measured and affords an index of the loss of plasma volume.

LOSS OF WATER AND ELECTROLYTES.—It is well recognized that dehydration associated with the loss of body fluids or with an inadequate intake of water and salt may lead to shock. Although initially there is a concentration of both the plasma proteins and the red blood cells, if the volume of circulating blood is sufficiently reduced to impair the blood supply to the tissues, a secondary loss of plasma protein probably occurs. Simple restoration of the fluid lost is not sufficient to restore the blood volume. The reduced level of the plasma proteins, after the administration of water and crystalloids, indicates that there was probably a secondary loss of protein from the blood stream during the period of dehydration.

Low Blood Pressure without Loss of Blood Volume. REFLEX FALLS OF BLOOD PRESSURE.—Just as hemorrhage may lead to a discrepancy between the blood volume and the capacity of the vascular bed, so an increase in the capacity of the latter may have the same effect. There results a drop in blood pressure with reduction in the distribution of blood to the peripheral tissues. The initial fall of blood pressure immediately after the receipt of an injury, which has been termed "primary shock," is probably of this nature. A similar condition is frequently observed to result from manipulation of the contents of the upper part of the abdomen during operations in this region. The exact mechanism of this decrease in blood pressure has not been ascertained. It probably results from reduction in the cardiac output, either from segregation of blood in the veins, especially in the splanchnic region, with resultant decrease in the return of blood to the right side of the heart or from direct inhibition of the heart. A comparable condition is seen in the ordinary "fainting spell." Although prompt recovery generally occurs spontaneously the circulatory dynamics may be seriously upset. Studies on the collapse induced by the inclined posture, together with the administration of nitrites, indicate that severe deprivation of circulation may be produced.

SPINAL ANESTHESIA.—The fall in blood pressure frequently observed to follow the administration of spinal anesthesia is again one which is not initially associated with a loss of blood volume. Although originally believed to be due to a dilatation of arterioles, it has recently been shown that it is probably more dependent on a decrease in the return flow of blood to the heart, possibly through the loss of muscle tone. An additional explanation may be in the paralysis of sympathetic nerves going to the heart. The lack of acceleration of the pulse in spite of a low blood pressure is one of the characteristic changes observed in this condition. The significance of low blood pressure, even without initial loss of blood volume, in the production of shock, is dependent on the severity and the length of reduction of blood supply to the tissues.

LOSS OF MUSCLE TONE; "VENOPRESSOR MECHANISM."—The tone of the voluntary muscles and the movements of the respiratory muscles are probably of importance in assisting the return of blood through the veins to the right side of the heart. Henderson expressed the belief that the carbon dioxide content of the blood is important in controlling the tone of the muscles. He has also pointed out that the intramuscular pressure falls after operations and in cases of shock. With the generalized reduction in circulation that occurs as the process of shock advances, the muscles lose their tone and the tissues become "soggy." The venous return is thus probably still further reduced. It is a question, however, of whether this reaction is not more one of the sustaining than one of the initiating factors in the production of shock.

Reflex Vasoconstriction—A severe reduction in peripheral blood flow leading to shock can be produced by the prolonged injection of large quantities of epinephrine. This work has been criticized on the grounds that the dosage of epinephrine employed was far greater than could possibly have been secreted by the suprarenal glands. While it is true that it is unlikely that suprarenal secretion is ever sufficiently great or prolonged enough to produce the reduction in circulation sufficient to cause shock, the question on which these experiments were designed to throw light was "Can vasoconstriction, produced by reflex stimulation of the sympathetic nervous system, result in a reduction in peripheral circulation sufficient to produce shock?" Epinephrine was chosen simply to produce vasoconstriction and that amount was selected which would reduce the peripheral blood flow to the desired extent. Under these circumstances, it has been shown that the classical picture of shock, with hemoconcentration, loss of blood volume, and the pathologic picture of congestion in the splanchnic region can be produced.

Reduced circulation due to reflex vasoconstriction as exemplified by the injection of massive doses of epinephrine has been considered in detail because it may furnish an explanation of the mechanism by which certain traumatic stimuli can produce shock or aggravate the condition if present. Three of these stimuli—cold, pain, and fear—will now be considered.

COLD.—The influence of cold on the development of shock has been fully appreciated. During the war of 1914–1918, the relationship between the incidence and severity of shock and the climatic conditions in the field were noted by all members of the shock teams. The application of warmth has become a well established part of the treatment of shock. Reduced circulation is well recognized as the normal physiologic reaction to cold. It is to this impairment of circulation that the harmful effects of cold are probably to be attributed. Not only is the circulation reduced by reflex vasoconstriction, but the direct effect of a decreased temperature of the blood in producing an increase in viscosity is probably of importance. On the other hand, the danger of the overzealous application of heat, especially when there is a discrepancy between the blood volume and the capacity of the vascular bed, should not be overlooked. This condition will be discussed in Chapter II, on treatment (page 139)

PAIN.—On the basis of clinical experience it is generally agreed that pain is a factor in the production of shock. It is extremely difficult, however to obtain confirmation of this clinical impression under the controlled conditions of the laboratory. Possibly the necessary use of anesthesia, in animal experimentation, so changes the physiologic reactions that shock cannot be produced by pain alone. Again, there is probably a difference between man and animals in the sensitivity to painful stimuli. Finally even under controlled conditions there are many factors which are contributing to produce shock and it is difficult to evaluate the significance of pain among them. It is not surprising, therefore, that the reports on the significance of the nervous factor in shock have been contradictory. Cannon and Bayliss, in their original experiments on limb trauma, found that section of the spinal cord in the lumbar region did not prevent the occurrence of shock. However their work was criticized on the ground that the significance of local loss of fluid was not appreciated. Numerous experiments have demonstrated that shock could not be produced simply by painful stimulation but, again, other experiments have demonstrated that the exclusion of painful stimuli apparently prevented the development of shock. In experiments which recently have been performed Freeman and his associates found that section of the spinal cord apparently modifies the development of shock from injury to the ex

trémities Loss of blood volume, however, greater than could be accounted for by the loss into the region of trauma, was observed.

In summary, it seems probable that pain is an important contributory factor in the development of shock. Since painful stimuli are known to result in a reduction in circulation, it is likely that their effect in the production of shock is mediated through tissue anoxia. The circulatory changes produced in patients by the perception of painful stimuli are identical with those that are recognized as an integral part of the process of shock.

FEAR, PSYCHIC INFLUENCES—The significance of fear or emotional disturbances in the production of shock is again a matter of clinical impression rather than of fact substantiated by controlled experimentation. Instances have been recounted in which profound shock, even resulting in death, has resulted from the receipt of trifling wounds. The emotional attitude of a patient before a major operation frequently has been felt to be a determining factor in the ultimate outcome. The mechanism through which fear exerts a deleterious influence is probably reflex stimulation of the sympathetic nervous system, comparable to that resulting from pain and bringing about such circulatory alterations as have been found to accompany the injection of massive doses of epinephrine. The extreme sympathetic hyperactivity associated with the "pseudoaffective" state in cats after removal of the superficial portions of the cerebral cortex has been found to result in shock, and the loss of blood volume attendant on this condition can be prevented by total sympathectomy. Those who have studied the effect of slight emotional stimulation on the peripheral blood flow, in both man and animals, have been constantly impressed by the dramatic and marked reductions in circulation which such stimulation can produce. The peripheral blood flow in one case of so-called psychic shock was found to be reduced for some period of time to a degree usually found in shock. Recovery with reassurance took place spontaneously. In recent experiments on traumatic shock with restriction of local loss of fluid, it was observed that the emotional reactions of the dogs appeared to be of significance in the incidence of shock. On one occasion, when a control experiment was being run, an extremely apprehensive dog was kept on the table for three hours without any trauma, simply in order to determine the effect of the experimental procedure on the circulation. The peripheral blood flow was severely reduced throughout the experiment. A slight degree of hemoconcentration occurred and a second determination of the blood volume by the carbon monoxide method showed a 20

per cent reduction in the blood volume, from that measured at the start of the experiment.

It seems likely therefore, that the emotional state may play a significant part in the production of shock. Objective proof of this concept, however is difficult to obtain.

Toxic Factor in Shock

One of the most attractive theories to explain the reduction in volume of circulating blood, from increased capillary permeability, as the mechanism of shock was the theory of traumatic toxemia. According to this idea, there was absorbed from traumatized tissue a toxin which was carried by the blood stream to all parts of the circulation, where it had a specific effect on the capillaries, causing an increase in their permeability and allowing the plasma to escape. The red blood cells were left stranded and, in this way, the pathologic picture of capillary congestion was produced. Experimental evidence advanced to support this concept was of four varieties (1) the administration of toxic agents, such as capillary poisons, injured tissues or extracts of such material, (2) experiments in which the absorption of the hypothetical toxin was temporarily prevented by the use of tourniquets, (3) cross-circulation experiments, and (4) experiments in which reduced circulation was excluded as an initiating factor. In addition, many unsuccessful attempts have been made to demonstrate toxic material in the circulating blood and lymph. It may be said that the evidence against the traumatic toxemia theory has been of a negative sort. The experiments supporting the concept have been criticized on the ground either that the conditions were not such as would be obtained in patients or that the resultant shock could be accounted for by other factors.

Toxic Agents. HISTAMINE AND PRODUCTS OF TISSUE INJURY—Since Dale, Laidlaw and Richards had demonstrated that the intravenous injection of histamine could produce shock, it was only natural to ascribe to histamine the role of the hypothetical toxin responsible for this condition after trauma. Although the intravenous injection of histamine produces widespread injury to the capillaries, leading to increase in their permeability with consequent loss of plasma, hemoconcentration, and a postmortem picture of congestion, it is unlikely that this substance is the hypothetical toxin responsible for the development of traumatic shock. Recent analyses have shown that the muscles are especially poor in their content of histamine, while it is recognized that traumatic shock is particularly likely to follow the crushing of large masses of muscle tissue. Again, the concentration

of histamine found in the blood is not necessarily elevated in shock. Perhaps the strongest evidence against the possibility that traumatic shock is due to the action of histamine is offered by the work of Dragstedt and Mead, who were unable to demonstrate any histamine-like substance in the blood and lymph from the thoracic duct of animals which were in a state of shock as a result of trauma. With the same technic they were able to demonstrate the presence of histamine in the blood and lymph in the presence of anaphylactic shock and after subcutaneous or intramuscular injection of histamine. Even though histamine seems to have been excluded as the toxic factor in shock there are many other products of tissue metabolism or breakdown which may be responsible. Viviperfusion experiments of Parsons and Phemister and the "vividialysis" experiments of O'Shaughnessy and Slome were unable to demonstrate any vasodilator or depressor activity in the blood, coming from the traumatized region, or in the circulating blood stream. On the other hand, the criteria used to determine the presence of a toxic substance was either a depression of blood pressure or vasodilatation. It is still conceivable that the toxic factor might have no influence on the tone of the larger vessels but still might have a specific effect on the capillaries.

POTASSIUM—The suggestion has been made by Scudder and his associates that an increase in the potassium content of the blood played a significant part in the production of shock. Their finding of a consistent elevation in the serum potassium has not been confirmed by other investigators. In addition, there is no evidence that an increase in the capillary permeability leading to a reduction of blood volume can be produced by alterations in the potassium content of the blood such as they have found.

SUPRARENAL CORTICAL HORMONE—That shock can be produced by removal of the suprarenal cortex has been fully proved. Again it has been clearly shown that animals deprived of their suprarenal cortical hormone are extremely sensitive to trauma and other shock-producing procedures. On the basis of these observations, the suggestion has been advanced by Swingle and his associates that traumatic shock was essentially the result of suprarenal cortical insufficiency and the treatment of shock by the administration of this hormone has been advocated. It was believed that the tone and permeability of the capillaries were directly influenced by the hormone. Although it is recognized that suprarenal cortical hormone is indispensable in the treatment of shock associated with suprarenal cortical deficiency, no convincing evidence has been advanced to support the view that

it is essential for the treatment of shock which occurs from trauma in the previously healthy subject. During the process of shock, however there is injury to a great variety of tissues as a result of the impairment of circulation. It is quite possible that the suprarenal cortex may suffer injury in this manner. If so, an additional factor contributing to the progressive loss of volume of circulating blood may be produced. It has been found that the duration of increased capillary permeability which followed severe burns of patients could be shortened by the use of suprarenal cortical hormone. It seems quite possible that the suprarenal cortex may play a significant part in the mechanism of shock under certain conditions.

TRANSPLANTATION OF INJURED TISSUE.—The intraperitoneal transplantation of traumatized tissue, liver skeletal muscle, burned skin, and so on, frequently has been used in the study of shock. Although occasional negative results have been obtained, the consensus is that a type of shock associated with severe hemoconcentration can be produced in this manner. However as Cannon and Bayliss have pointed out "A primary consideration in the experimental study of natural phenomena is that the reproduction of the phenomena under controllable conditions shall resemble as closely as possible the occurrences in nature." The placing of skeletal muscle or liver within the peritoneal cavity hardly duplicates conditions likely to be encountered in patients.

Tourniquet Experiments.—In their original experiments on traumatic shock, Cannon and Bayliss found that this condition could be prevented by the application of a tourniquet, above the region of trauma, sufficiently tightly to occlude the blood supply completely. On release of the tourniquet, shock soon occurred. They advanced this evidence in favor of the accumulation of a toxic material in the traumatized region which was released into the blood stream only after removal of the tourniquet. Their experiments were criticized by Blalock on the ground that insufficient attention was paid to loss of blood into the traumatized region after release of the tourniquet. "When the femoral artery was isolated in the groin and a tourniquet was placed tightly around the thigh, compressing all structures except the artery and preventing all venous or lymphatic return from the part, it was found that severe trauma to the extremity caused a decline in blood pressure and death. The possibility of the absorption of toxic products in these experiments was excluded and death was explainable on the basis of the local loss of fluid."

Release of a tourniquet which has rendered a limb ischemic for a prolonged period (three hours) is frequently followed by a fall in

blood pressure leading to shock. Wilson and Roome found that the ischemic extremities were grossly swollen and that the fluid which escaped from the tissues on incision contained a high concentration of protein. They attributed the shock produced in these animals to primary loss of volume of circulating blood into the region which had been injured by prolonged anoxia. They found, however, that the injection of very large quantities of either plasma or blood, or amputation of the legs at the level of the constriction alone, did not prevent death. However, when amputation was performed and the lost fluid replaced, two of their animals did recover. It seems possible that a dual mechanism was responsible for the shock in their experiments the loss of plasma into the injured region and the absorption of a toxic material.

Cross-Circulation Experiments—Convincing proof of the existence of a toxic factor in the production of shock would appear to be readily obtained by cross-circulation experiments. Experiments in which the traumatized limb has received its circulation from a second animal have given variable results. The difficulty of evaluating the part played by local loss of fluid has confused the interpretation of the results. Transfusion of blood from a shocked to a second animal has not resulted in a fall in blood pressure of the recipient. Recent studies by Best and Solandt on massive, equal, exchange transfusions between a shocked dog and a recipient have shown an occasional death of the test animal which could not be explained otherwise than through the passage of some toxic material from the shocked animal.

Exclusion of Reduced Circulation as Initiating Factor—The traumatic toxemia theory has suffered, as Blalock has pointed out, "by lack of proof that local injury in itself produced general damage to all capillaries and tissues by the elaboration of a toxic substance." As mentioned previously, either the conditions of the experiments were not comparable to those present in clinical cases or the production of shock could as well be explained on the basis of some other concept. Since it was felt that the loss of blood volume could be explained either through reduced circulation or through the action of some toxic factor, it seemed necessary, in order to evaluate the part played by this hypothetical toxic substance, to perform experiments in which the reduction in circulation could be excluded as an etiologic factor. To accomplish this end, it was first of all necessary to prevent excessive local loss of fluid into the traumatized region. This object was achieved through the application of bandage and adhesive tape to the extremities which were to be injured. It was again neces-

sary to prevent reflex vasoconstriction from producing a reduction in circulation. To accomplish this purpose, the dogs were subjected to total sympathectomy by previous aseptic operations. It was again important to ascertain whether a reduction in circulation had been

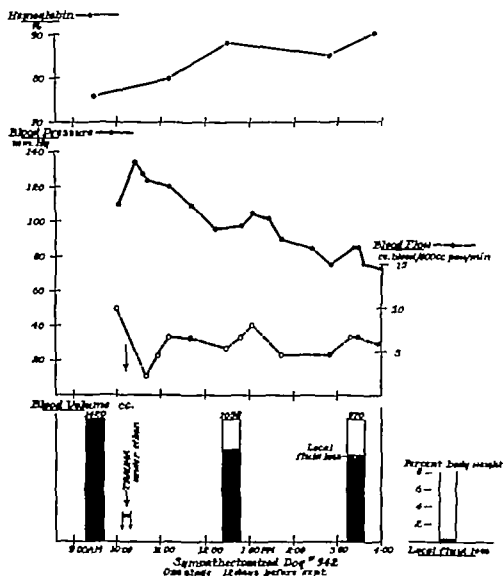


Fig. 3.—The effect of trauma under ether anesthesia on blood pressure, blood flow concentration of hemoglobin, and blood volume of a dog after total sympathectomy

prevented. Determinations of peripheral blood flow and the oxygen saturation of the venous blood from the right side of the heart indicated that, according to these criteria, the circulation was not seriously disturbed. As far as the criteria of shock were concerned, loss in volume of circulating blood, more than could be accounted for by

the local loss of fluid, was adopted. In addition, an increase in the concentration of the red blood cells and the demonstration of capillary congestion in the splanchnic region were used as indicators of an increase in capillary permeability. Under these conditions it was found, as is shown in Fig 3, that a decrease in volume of circulating blood could be produced by trauma. This lowering of the blood volume was greater than could be accounted for by the local loss of fluid into the region of injury. It was produced in spite of evidence of an adequate circulation. It was accompanied by hemoconcentration and evidences of congestion in the viscera. From these facts, together with the other evidence which has been reviewed, it seems probable that there is a factor in addition to reduced circulation which is significant in the production of shock.

SUMMARY

Shock is the ultimate result of the action of a number of different factors. In the first place, the general condition of the subject prior to the injury often plays a deciding part. Fatigue, dehydration, and exhaustion from physical and mental stress may have depleted his energies. The loss of blood, plasma, or body fluid occasioned by the injury produces a primary reduction in circulation. Reflex vasoconstriction in response to the loss of blood volume and to the traumatic stimuli, both physical and mental, brings about a further reduction in circulation. In addition, the absorption of some product of tissue breakdown which has a specific effect on the permeability of the capillaries throughout the body may contribute directly to loss of plasma volume. Once circulation has been reduced, the tissue anoxia which results from it causes progressive injury to the capillaries, allowing further loss of plasma volume. Hemoconcentration, with its attendant increase in viscosity, adds to the circulatory deficiency. Other sustaining factors are brought into play as the process of shock runs into its vicious cycle. Prompt interruption of this cycle, before irreversible changes have been produced in the tissues, is needed to bring about recovery. Such interruption can be effected by replacement of lost fluid and exclusion of traumatic stimuli and toxic factors.

CHAPTER II

PREVENTION AND TREATMENT OF SHOCK*

ALFRED BLALOCK, M.D

THE treatment of shock depends to some extent on the nature of the injury or disease responsible for its development, but certain forms of treatment are indicated regardless of the causes. It is apparent that removal of the cause is indicated if this is feasible. It is unfortunately true of many of the accidents in civil and military life that the damage has resulted in peripheral circulatory failure before attempts to combat its development have been possible. Efforts to prevent shock which may accompany various types of operations have been more successful.

Advances in the prevention of shock have more than kept pace with improvements in methods for treating the fully developed condition. After the blood pressure and blood volume have remained depressed for a considerable period, general damage to the tissues occurs as a result of the anoxia and no form of treatment results in sustained improvement. It is for this reason that the major effort should be directed toward the prevention of or the earliest possible treatment of, peripheral circulatory failure. The most effective form of therapy should be carried out with as little delay as possible.

General Condition of Patient

There is no doubt that the condition of the patient at the time of injury or operation is an important factor in determining the likelihood of the development of shock. Dehydration, acid-base disorders, and nutritional disturbances are among the many abnormalities which influence the likelihood of the development of shock. In the war of 1914-1918 it was observed that among soldiers whose fluids were adequate, shock did not develop following injury as readily as among those whose fluids were insufficient. Hypoproteinemia as a result of inadequate food or other factors causes a decrease in the osmotic pressure and an increase in the ease with which fluid is lost from the blood stream.

* Use of blood and blood substitutes not included in this chapter. See Chapter III.

The realization that active bleeding constitutes one of the few emergency conditions requiring immediate operation has meant much to the safety of patients. In the great majority of instances, the possibility of the ultimate recovery of patients who are in a state of incipient or fully developed shock is greatly strengthened by carrying out appropriate therapy prior to the performance of the operative procedure. This preliminary treatment is particularly important if means are not available for the intravenous administration of fluids, or blood, or both, during the operation. One of the conditions most frequently encountered in surgery is dehydration. The observations of Collier and others on this subject merit consideration.

Dehydration refers to a reduction in the volume of body water. This is a particularly serious complication or contributing factor in cases in which peripheral circulatory failure is present because shock usually results in inability of patients to retain and absorb fluids administered by mouth. Furthermore, fluids introduced subcutaneously or by rectum are absorbed very poorly under such circumstances.

Water is normally lost from the body by way of the kidneys, skin, lungs, and intestines. The amount that is put out in the urine varies with the amount that is available and with the waste products that are to be excreted. If the kidneys are capable of concentrating urine to a specific gravity of 1.030, 600 cc. of urine are required for the removal of waste products. In the presence of renal disease or damage, due to shock or other causes, larger quantities are necessary. Under normal conditions the loss of water in feces is usually not greater than 150 cc. daily but, in the presence of severe diarrhea, the loss may be very great. The remaining normal loss of fluid is that which is known as the insensible loss. This refers to the loss from the skin and the lungs. Even in the absence of sweating it has been found that a normal person loses 1000 to 1500 cc. of water daily through the lungs and the skin. This loss is continuous and takes precedence over that by the kidneys in the excretion of waste products. Conditions which lead to sweating, such as pain, exertion, and shock, result in a great increase in the loss of water.

The abnormal loss which accompanies diarrhea has been mentioned. One of the commonest causes of dehydration is the loss of fluid from the gastro-intestinal tract by vomiting. The total volume of intestinal secretions is surprisingly large, for an adult, it is approximately 8000 cc. daily. By comparison, the total volume of blood plasma is approximately 3500 cc. A high percentage of the intestinal secretions is normally reabsorbed, but this is not the case in the presence of persistent vomiting or diarrhea. These secretions are

formed, in the main, from the water and the electrolytes of the blood plasma. The electrolyte composition of the combined secretions is approximately the same as that of the blood plasma. It is apparent that the loss of these secretions by vomiting or diarrhea will upset the fluid balance of the body unless replacement therapy is carried out.

The fluid requirements of a healthy person are usually met by the taking of sufficient water to alleviate thirst and by the water contained in the food. This usual intake is about 3 liters per day: 2 liters is taken as drink, 0.5 liter in food, and the remainder as water of oxidation. If the subject is perspiring profusely, as is likely to be true of the soldier in combat, larger quantities of fluid are required. Many factors may play parts in the dehydration which may accompany injuries, diseases, or operations. Collier and Maddock have suggested a means for calculating the quantity of fluid that should be given to patients who cannot take fluid by mouth. This calculation is as follows:

1. Water for urine (24 hours)—1500 cc.
2. Water for vaporization (24 hours)—2000 cc.
3. Approximate replacement of fluid lost in vomitus, blood, feces, drainage from biliary and intestinal fistulas, exudation from inflamed surfaces—variable.

In addition, if the patient already is dehydrated

4. Water to restore depleted body fluids (6 per cent of body weight, estimated at 60 kg.)—3600 cc.

Even when the losses of fluid classed as abnormal are excluded, it is seen that at least 7 liters of fluid should be administered to the severely dehydrated patient. After the dehydration has been corrected, much less fluid is required.

The choice of fluid for replacement therapy is not always a simple matter. In the majority of surgical cases, profound dehydration is not present and fluids may be given parenterally simply because there is contraindication to their being allowed by mouth. Under these circumstances, the use of 5 per cent dextrose in distilled water is satisfactory. The problem is more difficult if severe dehydration is present but, at the same time, the employment of complicated solutions is usually not indicated. If renal function is adequate, and if water and sodium and chloride ion are supplied, the proper retention of these and of materials from metabolic processes will take place with resulting restoration of the extracellular fluids. In other words, the electrolytes can be replaced by the administration of physiologic solution of sodium chloride and renal function can be stimulated by

the giving of dextrose solution Means are available for determining the quantity of solution of sodium chloride that is required. If edema develops following the administration of solution of sodium chloride, its use should be alternated with that of dextrose

The statements thus far apply to the treatment of the patient who is dehydrated but who has not sustained severe *capillary damage* such as may accompany protracted diarrhea, prolonged intestinal distention, severe trauma to muscles, or burns Under these conditions the problem is much more complicated. Solutions of crystalloids are lost very readily and colloidal solutions, such as whole blood, plasma, or serum, should be administered It has been found that the giving of solutions of crystalloids, in the presence of capillary damage or of a fall in blood pressure from any cause, results not only in the escape of most of this fluid from the blood stream into the tissues, with resulting edema, but also in an additional loss of plasma protein.

It should be emphasized that there are many clearly established indications for the use of large quantities of solutions of sodium chloride and dextrose It must be remembered, however, that the effects of introducing noncolloidal solutions in the presence of a decided increase in capillary permeability are quite different from those obtained when the capillaries are normal Sufficient fluids should be given to correct the dehydration, but larger quantities may result in harm rather than benefit. When in the course of treatment it is found that the plasma protein is decreasing while the concentration of red blood cells is increasing, it can mean only that plasma is being lost from the blood stream and that the therapy is accomplishing little if any good

Acid-base disturbances will not be considered in detail. The treatment is frequently complicated by coexisting impairment of renal function due either to intrinsic disease of the urinary tract or to pre-renal causes, such as dehydration and peripheral circulatory failure attended or unattended by capillary damage. In the case of prerenal deviation without capillary damage, administration of saline and dextrose solutions will restore proper renal function as well as fluid balance with subsequent correction of moderate distortion of the electrolyte pattern of body fluids by renal activity. The presence of capillary damage may render such treatment ineffective The administration of large quantities of solutions of crystalloids, aiding in the transudation of protein from blood already deficient in colloid, may cause a further reduction in blood volume following transient hydremia, and there will be an accumulation of edema fluid rather than an increase in the urinary output. The desirable therapeutic

procedure is the transfusion of plasma or serum with the volume of solution for replacement limited to the quantity that is necessary to restore fluid volume.

Acid-base disturbances accompanied by or secondary to, true renal insufficiency are best combated by measures designed to bring about diuresis. Relative polyuria is necessary in order that the kidneys, deficient in concentrating power, may get rid of the end-products of metabolism. Here again the administration of saline and dextrose solutions is of the greatest benefit, permitting replacement of the base and fluid lost as well as supplying extra fluid with which the failing kidney can eliminate metabolic end products in low concentrations.

Food should be adequate in quantity as well as in composition, an abundance of protein is especially important. The ill effects that may result from hypoproteinemia are well known. A good state of nutrition increases the ability to withstand infections and injuries. The evidence indicates that adequate vitamins, particularly vitamin C, are of importance in the healing of wounds and in the combating of infections.

Exposure to cold, with inadequate clothing, increases the susceptibility to shock, particularly if the clothes are wet. Dry garments should be available for replacement and should be substituted for the wet ones as early as possible. In effecting this exchange, injured parts should be moved as little as possible.

First-aid Dressings

In addition to the control of hemorrhage and the splinting of fractures, there are a number of points of importance in first-aid treatment. It is obvious that a consideration of the general condition of the patient and attempts to combat shock take precedence over the treatment of the local injury provided the local loss of blood has been stopped, but the great importance of the proper care of wounds must not be minimized. The carefulness and promptness of the first aid treatment of open wounds have much to do with determining the likelihood of the subsequent development of severe infection. It is preferable to attempt no first aid treatment, and to leave a wound without dressing, than to do it carelessly.

If the general condition of the patient warrants, the wound should be adequately exposed. If indicated, the clothing should be cut rather than unbuttoned. It should be remembered that pathogenic organisms from human sources frequently gain entrance into the wound after rather than at the time of, injury. Unless profuse bleed

ing is taking place, wounds should be let alone except for covering them with a sterile dressing until the patient has been transported to a place where aseptic technic can be employed. An exception arises in the case of a sucking wound of the chest, then immediate closure by suture or by the use of adhesive tape is indicated. The method for removal of bacteria and other foreign materials from wounds should be *mechanical* rather than by the use of antiseptic substances. The important point is that wounds should be let alone except for the introduction of a sulfonamide and the application of a sterile dressing, provided the bleeding has ceased, until facilities are available for proper care. The wound should not be touched with unscrubbed fingers, unsterile dressings, or other unclean objects.

Experience may show it to be advisable to administer one of the chemotherapeutic agents locally and generally to all patients with open wounds.

Control of Hemorrhage

Hemostasis should be accomplished by measures which interfere as little as possible with the nutrition of the tissues. For this reason a tourniquet should not be applied to an injured extremity unless other means of controlling the bleeding are ineffective. Wilson and Roome found that removal of a tourniquet is followed by the passage of a considerable part of the blood into the dilated vessels of the extremity and frequently by a decline in blood pressure. If a tourniquet is found to be necessary, it should be applied tightly enough to interrupt the arterial inflow to the part. Occlusion of the venous return alone results in more harm than occlusion of both the arterial inflow and the venous return. Unless the part is almost totally destroyed, it is desirable that the tourniquet be released every hour in order to allow some blood to enter the extremity. The tourniquet should not be covered with a bandage and the patient should be instructed to tell every medical officer who sees him that he has on a tourniquet. In addition, the fact and time of the application of the tourniquet should be recorded on the patient's emergency medical tags. One of the best types of tourniquet consists of a blood-pressure cuff, but this is not usually available. If a standard tourniquet is not at hand, it may be necessary to improvise one from a belt, handkerchief, or necktie. It is advisable, particularly on the arm, to place a towel or some similar soft material beneath the tourniquet in order to prevent injury to nerves. If the means for doing such are available, the part distal to the tourniquet should be cooled.

As has been stated, a tourniquet should be employed only when

other means for controlling hemorrhage are inadequate. Other measures include the making of pressure on the artery between the wound and the heart, elevation of the extremity, application of a tight dressing, insertion of a sterile gauze pack or if adequate facilities are available, isolation of the vessel and the use of a clamp and ligature. In occasional cases it is necessary to ligate the artery proximal to the wound through a separate incision. The method that is used depends on a number of factors, including the nature of the wound and the facilities that are available for its care. It is unnecessary to emphasize further the importance of hemostasis. This is particularly true if the blood pressure has fallen to, or near, the critical level. Under such circumstances the loss of only a small additional quantity of blood may result disastrously. Every effort should be made to prevent unnecessary loss of blood.

Relief of Pain

There is evidence that *morphine* in moderate doses is of aid in the prevention of shock. It should be given for the pain and restlessness associated with injuries, except severe intracranial injuries, before the patient has been moved from the scene. Since it is possible to administer too much morphine and to add to anoxia already present in shock, usually not more than $\frac{1}{2}$ grain (0.032 gm.) should be given to the severely injured person. Phenobarbital sodium in moderate amounts may be given for the restlessness associated with intracranial injuries.

A possible valuable therapeutic aid in the prevention of shock may be found in the use of *sodium amytal*. The evidence on this point is not convincing and administration of the drug as a routine is not advised. Another possible aid in the prevention of shock following injuries of the legs may lie in the production of *spinal anesthesia*, as suggested by O'Shaughnessy and Slome. This procedure would appear to be inadvisable, however, as spinal anesthesia alone frequently is accompanied by a marked decline in the blood pressure.

Immobilization of Fractures

One of the greatest advances in surgery that resulted from the war of 1914-1918 consists in improvements in methods of dealing with fractures. Fractures of the long bones should be immobilized by the best means available before the patient is transferred from the scene of the injury. In addition to the reduction of pain, immobilization results in lessening of damage to the soft tissues, including blood vessels, by the sharp bony fragments, which is particularly impor

tant in lessening the further loss of blood. The *Thomas splint* for leg or arm is ideal for the splinting of fractures but is not always immediately available. Regimental surgeons should be provided with an adequate number of splints. Many common materials, such as pieces of lumber, make a poor substitute for temporary use. If substitutes for splints must be used they should be padded well on the side that is next to the skin and they should be bound securely by bandaging or by tying above and below the point of fracture, but not directly over it. Fractures of the thigh are particularly difficult to treat by improvised means since it is important that traction be applied. Every effort should be made to obtain the proper type of splint before the patient is transported any considerable distance. The future will witness an increase in the use of wires and pins for ensuring fixation and traction.

Infiltration anesthesia has been found to be satisfactory for use in reduction of fractures, particularly those about the ankle and wrist. The needle is inserted directly into the hematoma and the solution is injected. Satisfactory anesthesia usually can be obtained within five to ten minutes by injecting 10 to 20 cc. of 1 per cent solution of procaine. This method should not be used in dealing with compound fractures.

Immobilization for Other Injuries

The results of recent investigations point to the value of immobilization for all severe injuries of the extremities, whether fractures are present or not. The benefit may be due to prevention of the spread of infection by lymphatic structures, reduction in the absorption of toxins, lessening of the local loss of fluid, or to other causes. At any rate, extremities which are severely injured should be immobilized.

Rest and Quiet

Although there is a difference of opinion as to the manner in which rest and quiet exert their beneficial effects, it is agreed that they are helpful in the prevention and treatment of shock. Undertreatment rather than overtreatment is of more frequent occurrence, but overtreatment is a reality in some instances and one should not forget that the patient needs rest.

Heat

A definite relationship exists between the incidence of shock and the loss of body heat, and an effort should be made to prevent and to correct chilling. During the examination and treatment the injured

man should be subjected to as little exposure of the body as possible. When possible, preliminary dressing of wounds should be performed in a warm place. Heat is lost especially rapidly through wet clothing, and dry garments should be substituted as early as possible. All ambulances should be equipped with heating devices.

A number of devices may be used for supplying heat. If *blankets* are at hand, they should be placed around the patient whether a bed is available or not. As Cannon has emphasized, more blankets are needed under the body than over it. The reason is that the blanket protects against loss of heat by the air which it holds enmeshed in its fibers. The weight of the body lessens the air space in the fabric and reduces the amount of protection from the undersurface. Since there is usually a scarcity of blankets in time of war, they should be used as efficiently as possible.

There are various other means for supplying heat. These include the use of canteens or bottles filled with hot water and of warm stones or bricks, which should be placed outside the clothing in order to prevent burns and between two surfaces such as the legs or the arms and the thorax. In addition, the patient should be given hot drinks unless there are contraindications such as a penetrating wound of the abdomen. In addition to supplying heat the fluid supplements the reduced body fluids. If fluid cannot be retained when given by mouth, as is often the case in the severely wounded, warm liquids may be given per rectum. Great improvement in the condition of the cold, wounded patient may occur when he is placed in a warm bed. It may be possible to place electric lights under a cradle which is over the bed, or a lantern can be placed on the floor beneath the bed or stretcher which should be dressed with blankets to provide for retention of warm air beneath.

It should be borne in mind that excessive heat may exert as ill effects as excessive cold. If patients are in the condition of secondary (hematogenic) shock there is a diminution in blood volume and vasoconstriction. The extremities are cold, but this is due in part to the fact that the blood is diverted in the main to the more vital structures, such as the heart and the brain. Excessive heat may cause dilatation of the vessels of the surface of the body and the flow through the more vital parts will suffer correspondingly. Cold extremities may be associated with a definite elevation of the internal temperature and excessive heat may have ill effects. Further more, the patient who is in a condition of shock is usually dehydrated and the application of heat, with the resulting sweating, will exaggerate the condition. These statements are in no sense a criti

cism of the very important principle of keeping the patient warm, but a considerable elevation of the skin temperature should not be caused unless, at the same time, the reduced volume of blood is supplemented by introducing blood or blood substitutes

Shock Position

It is fairly generally agreed that the so-called shock position, in which the foot of the bed is elevated, results in some improvement in the presence of peripheral circulatory failure. Experimental observations during the war of 1914-1918 did not present any convincing evidence of benefit. It is possible, however, that lowering the patient's head increases the flow of blood to the brain. It is well known that nerve tissues withstand a poor blood supply less well than others. Another theoretical benefit that might be derived from this position is in connection with facilitation of the venous return from the large abdominal vascular region. However, since most of the blood is not in the large veins but elsewhere, it seems doubtful if the position is important in this respect. There is no proof that the blood of the portal vein can be made to pass through the hepatic capillaries by gravity drainage. Wells and his associates recently presented evidence that lowering the head and flexing the neck on the thorax increase the cardiac output and improve the condition of the animal that is in a state of shock. It is probably a sound impression that the shock position should be part of the therapy of patients in shock.

Drugs That Cause Vasoconstriction

A number of vasoconstricting drugs have been recommended for the treatment of shock. Included among these are epinephrine, ephedrine, caffeine, camphor, neosynephrin hydrochloride, ether, strychnine, 25 per cent solution of pyridine betacarboxylic acid diethylamide (coramine), metrazol, pitressin, and benzedrine. As stated previously, however, vasodilatation is rarely present in association with secondary shock, except in the terminal stages, and induction of further vasoconstriction in early stages may result in harm rather than benefit. The elevation in pressure which follows the injection of drugs such as those named results from increase of resistance in the tips of the arterial tree. As Cannon has stated, damming the blood in the arterial portion of the circulatory system at a time when the organism is suffering primarily from a diminished quantity of blood does not improve the volume flow in the capillaries. Merely a higher arterial pressure is not the aim in the treat-

ment of shock, but rather a higher pressure which transmits an increased flow of blood through the capillaries all over the body. This result can be accomplished in the presence of secondary shock only by increasing the blood volume. On the other hand, some patients who are in a state of shock present a combination of reflex vasodilatation as well as a reduction in blood volume, and the use of vasoconstricting drugs may result in benefit. In the presence of shock due to uncomplicated loss of blood, it may be advisable, as suggested by Best, to give one of these drugs while plans are being made for the injection of blood or a blood substitute.

In primary or neurogenic shock, which is usually of only short duration, the use of vasoconstricting drugs may be indicated. For example, the vasodilatation and the decline in blood pressure which may be associated with the production of spinal anesthesia should be treated by the injection of a vasoconstricting drug such as ephedrine.

In general, it can be stated that the employment of vasoconstricting drugs is rarely indicated in the treatment of traumatic shock.

Inhalation of High Concentrations of Oxygen

The oxygen content of the arterial blood usually is essentially normal in cases of shock but that of the venous blood is greatly reduced (stagnant anoxia). The inhalation of 100 per cent oxygen results not only in an increase in the amount of oxygen in chemical combination with hemoglobin, but also in a substantial increase in the amount of oxygen in physical solution in plasma. There is a resulting increase in the pressure of oxygen in the tissues. High concentrations of oxygen can be more effectively and cheaply administered by use of the B.L.B. mask than by any other available means. If the respirations are depressed, carbon dioxide should be added to the oxygen.

Although there are a good many theoretical and practical reasons for the administration of oxygen in shock, the results of such treatment are not striking. During the exigencies of warfare, it is doubtful if the benefit that will result will be worth the time and effort. The transportation of large tanks of oxygen presents a difficult problem. Certainly the routine administration of oxygen in cases of shock should not be carried out if it interferes with some of the more necessary and more beneficial procedures.

Suprarenal Cortical Extract

Points of similarity between shock and Addison's disease have suggested the use of suprarenal cortical extract in the treatment of

shock. Potent extracts have been available only in recent years and it is too early as yet to assess the value of this form of therapy. Possible beneficial effects may result from decreasing abnormal capillary permeability or by reducing hyperpotassemia. Scudder suggested that the use of cortical extract should be combined with that of hypertonic solution of sodium chloride. From the evidence presented thus far, one is not justified in rating suprarenal cortical extract as one of the several most valuable agencies in the treatment of shock. Some of the reports are encouraging, particularly those from Canada, but definite proof of benefit in the treatment of traumatic shock is lacking.

Anesthesia

Opinions on this subject undoubtedly vary rather widely. Included among the problems presented by the wounded person in relation to anesthesia are his general condition, the type of wound, the part of the body which is injured, and the length of time between the injury and the operation. Among the important factors which enter into the choice of anesthetic agents are bulk and weight, transportability, explosibility, and fire hazard, as well as physiopathologic effects of the agents themselves.

In the war of 1914-1918, a great deal of enthusiasm for *nitrous oxide* was expressed. This was due in part to the observation that animals anesthetized with nitrous oxide and oxygen were more resistant to histamine than those anesthetized with ether. It is appreciated at present that surgical anesthesia cannot be produced with nitrous oxide without causing lack of tissue oxygen, anoxic anoxia. Stagnant anoxia is already present in cases of shock and it is unwise to cause additional deprivation of oxygen. Certainly, nitrous oxide, with oxygen, should not be used as the sole agent in prolonged operations. Nitrous oxide is of advantage in that it is nonexplosive, but transportation of the large cylinders is a difficult problem. It would seem unwise to attempt to use it in front-line stations.

Cyclopropane is a satisfactory agent for operations on patients who are in a condition of shock since it can be used with a very high concentration of oxygen and it exerts a minimum of toxic effects. Beecher has found recently that it is the best anesthetic agent for inhalation in the presence of experimental traumatic shock. The greatest drawbacks to its use are its high explosibility, the difficulty of transportation, as with all gaseous anesthetic agents, and the fact that expert administration is required. Ethylene presents no great advantages or disadvantages as compared with cyclopropane.

Ether is the safest of the anesthetic agents for inhalation. It is likely that its harmful effects in the presence of shock, except for prolonged procedures, have received too much emphasis. When combined with a high concentration of oxygen it is among the better agents. The mixture of oxygen and ether is explosive, however, and the bulk of the oxygen makes for difficulty of transportation. Even without oxygen, ether continues to be one of the most valuable agents and most physicians are able to administer it.

Spinal anesthesia with nupercaine, procaine, and other agents is ideal for operations on the lower extremities and lower part of the abdomen if blood volume and blood pressure are not significantly depressed. These agents cause vasodilatation and thus renders less effective the reduced volume of blood. The stagnant anoxia of spinal anesthesia is added to the stagnant anoxia of shock. The tolerance of hemorrhage is less under spinal anesthesia than under ether. Spinal anesthesia is not contraindicated, however particularly if vasoconstricting drugs are available, unless circulatory failure is of significant degree. The advantages presented by spinal anesthesia include ease of administration of the agent, ease of recovery with little need for nursing care, and the small bulk of the agents.

Since the war of 1914-1918 real advances have been made in *intravenous anesthesia*. A number of short acting barbiturates are available and, by intermittent administration as the need arises, they can be given with very little danger. If given in small amounts repeatedly, their administration is not contraindicated in the presence of shock. One should avoid large doses, which cause histotoxic anoxia. The best of the short-acting agents at present are pentothal sodium and the sodium salt of *n*-methyl-*C-C*-cyclohexamylmethyl barbituric acid (evipal soluble). Lundy's method of intermittent administration should be used. In the employment of pentothal sodium the solution (2.5 per cent) is drawn into a 20 cc. syringe. The needle is introduced into a vein and is held there by the anesthetist. After the injection of 3 to 4 cc. of the solution the patient is instructed to count slowly. When the patient stops counting, the surgeon may begin the operation. With the needle remaining in the vein, the drug is injected as is indicated in order to maintain adequate anesthesia. It is necessary at present that an attendant should be at hand in order to be certain that an open airway is maintained. Agents for intravenous use have the advantage in warfare of small bulk and weight and of presenting no fire or explosion hazard. It is a safe prediction that their use will increase, although this point of view is not supported by some of the early experience in the present war.

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Cyclopropane is a satisfactory agent for operations on patients who are in a condition of shock since it can be used with a very high concentration of oxygen and it exerts a minimum of toxic effects. Beecher has found recently that it is the best anesthetic agent for inhalation in the presence of experimental traumatic shock. The greatest drawbacks to its use are its high explosibility, the difficulty of transportation, as with all gaseous anesthetic agents, and the fact that expert administration is required. Ethylene presents no great advantages or disadvantages as compared with cyclopropane.

Local anesthesia is ideal for operations on patients who are in a state of shock and should be used whenever it meets the requirements. Nupercaine presents the advantage over procaine of producing surface anesthesia on application to mucous membranes, but procaine is somewhat safer for infiltration. Infiltration anesthesia is very satisfactory for the reduction of many noncompound fractures. When injected into the periarticular region it is also of value in lessening the pain associated with sprains. It should be of value in military zones.

If other agents are available, *chloroform* never should be used. Its only advantages are that it is not explosive and that only a small quantity of the agent is required. Tribromethyl alcohol (avertin), given by rectum, is dangerous because the rate of absorption varies greatly, furthermore, its prolonged action makes its employment inadvisable in the presence of shock.

In considering all factors it would appear that the *choice of agents* in the front-line stations may be limited to nupercaine or procaine (local, spinal), pentothal (intravenous), and ether. The same agents will suffice in base hospitals but it would be better to have cyclopropane available, and in any case to have oxygen which may be used in conjunction with ether or cyclopropane. For intrathoracic procedures the best agent is the mixture of oxygen and ether or of oxygen and cyclopropane administered through an intratracheal catheter. Spinal anesthesia is contraindicated for patients who are in a state of shock if operation on the thorax is necessary. Thus it is evident that even the base hospitals can function satisfactorily from the anesthetic viewpoint with only procaine, pentothal, and the mixture of oxygen and ether. Anesthesia teams should be organized and trained for duty in combat zones. It is possible that their functions should also include intravenous therapy.

Time of Operation

There are many reasons for early operations on wounded persons; among these reasons are, particularly, bleeding which cannot be stopped by other means and penetrating wounds of hollow organs such as the intestinal tract. As has been stated, the length of time separating the injury and the operation is an important factor in determining whether wounds should be closed by primary suture and whether tendons should be sutured, and whether shock and other complications will develop following penetrating wounds. In general, it can be stated that the mortality rate varies directly with the hours

intervening between the major injury and the operation, however, this statement does not imply that an immediate operation is indicated if the patient is obviously in too poor condition to tolerate the procedure. An attempt should be made to improve the general condition of the patient by the use of simple measures, such as administration of fluids, application of heat, and rest. The depressed blood volume should be supplemented by the intravenous introduction of blood plasma or whole blood. If the blood pressure has already fallen to the critical level (70 to 80 mm. of mercury systolic), it must be realized that the condition is very grave, that only a little additional trauma or loss of blood will result in profound shock, and that intelligent therapy is required. When the patient who is in poor condition is made ready for the operation, long and complicated operative procedures should be avoided and the principal lesion should be treated as quickly as the correct type of surgical technic will allow. At the same time body heat should be preserved, every effort should be made to prevent further loss of blood, the tissues should be handled as gently as possible, and very profound anesthesia should be avoided. In other words, careful surgery is indicated but reasonable haste is important.

Penetrating Wounds

Penetrating wounds do not form a part of this consideration except for the fact that their improper treatment is likely to lead to shock. Several dogmatic statements, to which there are undoubtedly exceptions, will be made in regard to the surgical care of patients with such wounds.

All penetrating wounds of the brain constitute acute surgical emergencies and should be treated by early removal of badly damaged tissue, including devitalized fragments of bone and those foreign bodies the removal of which will not increase the damage already sustained by the brain. If facilities are available, the irrigation-suction technic should be used. Local anesthesia, with epinephrine added to the anesthetic solution, is usually adequate.

As has been stated, sucking wounds of the thorax should be closed at the earliest possible moment. If injury to the heart and great vessels can be excluded, and if foreign material such as dirty clothing has not been carried into the thorax by the agent causing the injury penetrating wounds of the thorax usually should be treated by nonoperative means. If the wound is in the left lower part of the thorax, in the region of the diaphragm, abdominal exploration is in-

indicated because of possible penetration of the stomach or of the splenic flexure of the colon. If the wound is in the right lower part of the thorax, in the region of the diaphragm, exploration is usually not indicated. If the site and nature of the injury suggest damage to the heart, evidence of cardiac tamponade should be sought for. The two best evidences of cardiac tamponade are (1) suppression or absence of pulsation of the heart under the fluoroscope and (2) prominence of the veins and increase in the venous pressure.

All are agreed that in the presence of penetrating abdominal wounds exploration is indicated as early as feasible. Perforations of the intestinal tract should be closed, or if that is impossible, resection should be performed. Intestinal distention, which may lead to shock, should be prevented or treated by the employment of duodenal suction, intestinal intubation and inhalation of high concentrations of oxygen. Wounds of the urinary bladder should be closed and a catheter inserted. If the spleen is badly lacerated, it should be removed. Unless a kidney is too badly damaged, it should not be removed.

Prevention and Treatment of Complications

No attempt will be made to consider in detail the various complications which may arise in the treatment of surgical patients and which may contribute to the development of shock. Reference will be made only to intestinal distention and to pulmonary complications.

Intestinal distention interferes in varying degrees with the flow of blood in the vessels of the intestinal wall. The alterations may range from moderate venous stasis, with transudation of fluid into the lumen of the intestine, to necrosis of the intestinal wall. It has been found by Fine and others that great distention is associated with progressive diminution in the volume of blood plasma, greater than can be accounted for by the local loss.

Points of importance in the prevention and treatment of distention include restriction of ingestion of fermentable substances such as sugar and fruit juices, avoidance of cathartics, frequent alteration of the position of the patient in bed, application of heat to the abdomen, inhalation of high concentrations of oxygen, intelligent use of morphine and pituitrin, and employment of duodenal suction and intestinal intubation. The last two procedures are particularly effective.

Pulmonary complications may constitute other contributing agencies in the development of shock. Stagnant anoxia is usually present in cases of shock and the development of a pulmonary complication may superimpose anoxic anoxia. Points of importance in

prevention and treatment include avoidance of profound anesthesia, the Trendelenburg position during operations unless it is contraindicated, postural drainage, frequent postoperative alteration of the position of the patient, and intratracheal suction or bronchoscopic aspiration when indicated.

shock. In cases of late and severe shock, however, the superficial veins are frequently collapsed, and the circulation may be so poor that they do not fill when compression is applied above the point selected for insertion of a needle. Under such conditions it may be necessary to cut down on the vein in order to insert a needle or cannula. Venoclysis should be established at the earliest possible moment in all cases in which shock or potentially initiating factors of shock are present.

Although usually it is not necessary to cut down on veins to perform venoclysis, a simple, sterilized surgical kit for exposing a vein must be regarded as indispensable equipment in all places where patients are likely to be treated for shock. The kit should contain the following

- 3 towels
- 1 scalpel
- 1 dissecting scissors
- 1 tissue forceps
- 2 finely pointed hemostats
- 1 syringe of a capacity of 5 cc., with fine hypodermic needle
- 1 per cent procaine solution
- ligature material

The apparatus is completed by

- tincture of iodine
- 70 per cent alcohol
- small gauze sponges
- 1 roll of $\frac{1}{2}$ inch (about 15 cm) adhesive tape
- 1 padded arm splint
- 1 roll of $2\frac{1}{2}$ inch (about 6 cm) bandage
- 1 rubber tourniquet

The following technic is recommended.

The anatomic course of superficial veins varies widely, but there are certain regions, shown in Fig 4, where they generally can be reached.

Large superficial veins with heavy walls can be found (1) in the crease of the elbow, (2) over the lower end of the radius (accompanied by a branch of the radial nerve), (3) over the back of the hand, (4) over the dorsum of the foot, and (5) over the anterior aspect of the medial malleolus.

Compression of the vein is made with the tourniquet

After preparation of the field with iodine and alcohol, sterile towels are laid above and below the area. The skin is infiltrated with 1 per cent solution of procaine and a transverse incision is made. The incision is deepened, mostly by blunt dissection, until the vein is disclosed. The points of a hemostat are inserted close beside it, and the vein is freed from the surrounding connective tissue. The vein is then grasped with forceps and the hemostat thrust beneath it. A ligature is drawn beneath the vein and the hemostat again is

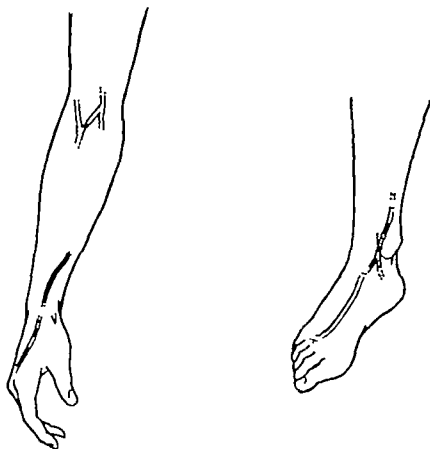


Fig. 4.—Regions where veins usually are accessible.

thrust beneath it so as to place the vein on tension. The ligature is tied distally

A second ligature is drawn beneath the vein. Place both ligatures under the vein simultaneously. The anterior wall of the vein is incised with scissors or a scalpel. The tip of the triangular flap is grasped with the forceps and the point of a needle or cannula is inserted into the lumen of the vein. The second ligature is then tied about the vein and the needle or cannula, and the intravenous infusion is started. One or more silk or catgut ligatures are used to

close the incision in the skin. A small dressing is applied to the wound, and the hub of the needle or cannula is fixed to the skin with adhesive tape. It is well, in addition, to fasten the rubber tubing, which lies proximal to the needle or cannula, either to the skin or to the splint to prevent accidental dislodgment. If a needle is used, the responsible person should be sure that the bevel opening is so oriented as not to be readily closed by pressure against the wall of the vein.

When the crease of the elbow or the wrist or the back of the hand is used, a padded splint is very desirable and, if patients are restless, unconscious, or delirious, indispensable.

Before initiating any operative procedure on a patient who is likely to go into a state of shock by reason of expected hemorrhage, prolonged anesthesia (especially spinal), extensive intestinal manipulation, débridement, and so forth, a needle or cannula should be placed in a vein, preferably of the foot or ankle. Glucose 5 per cent in physiologic solution of sodium chloride, should be used to maintain the needle open (1 to 2 cc per minute are sufficient). In case shock develops, at a moment's notice blood or plasma can be substituted. Best of all, a preventive dose of plasma or blood should be given to all such patients, and the needle or cannula should be maintained in the vein and should be kept open with glucose and sodium chloride solution. Then additional doses of plasma or blood can be given if necessity arises. Care must be taken not to "flood" the patient's circulation with excessive amounts of solution of crystalloids.

When the infusion has been terminated, the needle can simply be withdrawn. In case a cannula with enlarged tip has been used, it may be necessary to draw the tip against a sterile scalpel in order to cut the ligature. Simple pressure with a dressing usually controls the retrograde bleeding from the vein.

WHOLE BLOOD

For the past twenty years whole blood has been the most commonly employed material for reestablishment of proper blood volume in cases of shock.

The advantage of the use of whole blood in the treatment of shock is that it contains red cells, which is of advantage when severe anemia is present. This advantage, however, is more than balanced by many disadvantages, three of which are of great importance: (1) An interval of time is necessary to make whole blood available for transfusion, unless universal donor's blood is employed. (2) Preserva-

tion can be for a relatively short period and transportation is difficult because of the need of bulky and heavy apparatus for proper refrigeration. (3) The addition of red cells to the blood stream is often undesirable, especially when large quantities of blood are necessary (1000 cc. or more) or when hemoconcentration already exists. Even if hemoconcentration is initially not severe, addition of 2000 or 3000 cc. of whole blood would not be tolerated in the majority of cases, unless loss of blood had been severe.

The first and second objections are technical, and would be particularly operative in emergencies on the field. The third is one that is of especial importance in cases of late, severe shock and in the presence of burns.

The employment of citrated blood preserved by refrigeration is very widespread today. The useful period of safe preservation is relatively short. This period can be lengthened by the addition of glucose to the sodium citrate solution, a method first introduced by Rous and Turner. However the longer the period of preservation, the greater the danger of massive hemolysis from shaking during transportation. The Rous-Turner mixture, modified by DeGowin, Harris, and Plase, consists of two volumes of 3.2 per cent sodium citrate solution and thirteen volumes of 5.4 per cent solution of dextrose. Fifteen volumes of this solution are mixed with ten volumes of whole blood. In this mixture the rate of hemolysis is greatly retarded. However, the considerable dilution of the plasma proteins modifies the effectiveness of this mixture in the treatment of severe, late shock (see report of Case 5).

Even when a blood donor of suitable type is available, or refrigerated blood is on hand, at least one hour is required for proper cross-matching (collection of specimen from donor and patient, separation of serum, cross-matching, and the like). This loss of time can be avoided by obtaining blood from universal donors only, and possibly treating it with substances capable of neutralizing the A and B agglutinins. The limitations of this method are obvious. In addition to these drawbacks, the occurrence of post transfusion reactions must be considered. Generally mild and moderate reactions occur in from 5 to 10 per cent of cases. Severe reactions, usually due to hemolysis, are relatively rare and the incidence of death directly attributable to blood transfusion in the United States is variously placed at from 0.13 per cent by Tiber to 0.2 per cent by DeGowin. In the presence of shock, however, owing to the emergency to the temptation to hasten the preliminary steps in the preparation of blood and to increase the speed of administration, reactions are

more common. Another reason for increased incidence of post-transfusion reactions in the presence of shock is the fact that even in moderately severe cases, transfusions of large amounts (1000 to 2000 cc. and more) are necessary to obtain satisfactory results. Finally, it must be pointed out that it is not possible to administer blood safely at the very rapid rate often required by the critical condition of patients who are in a state of shock. Greater speed of injection is possible with other fluids, such as plasma.

These drawbacks in the use of whole blood in the treatment of shock are not enumerated in order to discourage the use of transfusion of whole blood, but rather to point out its relatively limited field of usefulness, particularly in the treatment of shock when emergency requires attention to large numbers of casualties on the field. It is to be regretted that lately transfusions of whole blood in many institutions have been neglected even more than justifiable limitations would warrant. Strictly speaking, however, in the emergency treatment of shock, whole blood is necessary only for the relatively limited number of patients who are in a state of shock accompanied by severe, *continued* loss of blood.

The preservation of citrated blood by refrigeration and the establishment of blood banks have received, lately, a great deal of attention and the literature on the subject is large and recent enough not to require repetition. Containers suitable for collection and transportation of citrated blood have been suitably described. Whatever the choice, the container must be such that it can be used either for administration of whole blood or for centrifugation and separation of plasma, so that the latter can readily be separated on expiration of the time of useful preservation of whole blood. It must be emphasized at this point that while a blood bank is not generally a convenient addition to small units, a plasma bank is always operable and of great service in a hospital of any size. In the organization of blood banks, therefore, provision must be made for the ready conversion of the whole blood into citrated plasma, at the expiration of the period of useful preservation of whole blood. For obvious reasons these considerations apply only to fixed, continental units of the armed forces.

To facilitate transfusion of whole blood in the field, it is essential to have: (1) each man properly typed, with the type clearly indicated on the identification tag; (2) part of the medical personnel properly trained in procedures for proper choice of the donor, as well as in the technic of collection and administration.

The most necessary element of any system for collection of blood for transfusion or for preparation of plasma is that it must be "closed"

to regulate effectively the volume of circulating blood. While it is agreed that the action of plasma and serum is similar, the former appears preferable because: (1) Plasma is easily obtained, often as a by-product of the blood bank. (2) Plasma gives a greater yield of the fluid phase (3) Extensive clinical experience of many investigators has shown absolute freedom from reactions, when the plasma is properly prepared and preserved, save for mild urticaria (4) Plasma contains more of the essential elements of whole blood.

On the other hand, *fresh* serum, or serum preserved by drying from the frozen state, produces reactions in a certain percentage of cases, with chill, fever, a sensation of tightness in the thorax, and so forth.

The primary toxicity of serum recently has been confirmed by the work of Scudder. Skin tests have shown that this toxicity decreases with storage in the liquid state. Such decrease probably explains the difference in results in the use of serum obtained by various investigators. Whether the action is due to thrombin remains to be confirmed.

Preparation and Preservation of Plasma—The problem of preservation of plasma is an important one and will be briefly outlined.

It has been stated elsewhere that, since the effect of plasma depends on the action of certain elements contained therein, and since these elements are variously affected by the manner of preservation, any method of preservation applied to plasma of human origin will profoundly influence its therapeutic value. In the use of plasma or of fluid given intravenously in the restoration of lost blood volume in the treatment of shock, first consideration must be given to the total protein content; secondarily, to its content of complement, because of its beneficial effect in combating the bacterial invasion that is so common in traumatic injuries. Relatively minor roles are played by fibrinogen, prothrombin, and the specific antibodies.

In considering the value of any therapeutic agent, first consideration must be given to the possibility of harmful effects on the patient. If plasma is separated with ordinary aseptic precautions from separate samples of citrated human blood immediately after collection from several patients, and if the plasma is then pooled and is administered at once to a patient, reactions are not encountered, save for mild urticaria. The routine use of this strictly fresh plasma, however, is not practical.

In the development of methods of preservation of plasma, it must be kept in mind that the safety of the material may be affected by (1) the comparative ease of bacterial contamination, with development of pyrogenic substances. (2) the tendency to flocculate of cer-

tain unstable globulins, particularly fibrinogen. Bacterial contamination is responsible for most of the febrile reactions which have been reported following administration of plasma.

Recently the report of a sudden death in the course of intravenous administration of unfiltered plasma has emphasized the absolute necessity of strict care in the prevention of massive flocculation or the imperative need of filtration to remove flocculi already formed.

Both bacterial contamination and flocculation can be effectively prevented, by rapid separation of plasma from freshly collected blood, followed by prompt fixation of plasma by freezing. Plasma can then be stored in the frozen state at minus 15° to minus 20° C. or, if it is considered desirable, it can be dried from the frozen state. A strictly "closed method" must be used as much as possible in these operations. While in the majority of cases plasma is a by product of the blood bank, it must be stated that storing of blood prior to separation of plasma for any length of time over the minimum that is necessary is to be avoided as much as possible. In the procedure of choice, plasma is separated and fixed by freezing within a period of from twenty four to forty-two hours from the time of collection of whole blood. In different institutions the period will vary according to the number of specimens collected, the hour of collection, and the procedure employed for the serologic diagnosis of syphilis.

Plasma "fixed" within this short period of time has been shown by clinical experience and by results of careful bacteriologic studies to be absolutely safe. In addition to bacteriologic studies, by both aerobic and anaerobic cultures incubated for five to seven days, each batch of plasma should be tested for toxicity by intraperitoneal injection into a white mouse.

Proper restoration to the liquid state of frozen or dried plasma results in a liquid free from flocculi. It must be noted, however, that while plasma properly restored from the frozen state is practically indistinguishable from the fresh material, plasma restored from the dried state is turbid. This turbidity however apparently causes no untoward effects.

To complete the measures to be taken to insure a uniform and safe material, provision should be made to pool eight to twelve lots of plasma, thus reducing an occasional high-titer iso-agglutinin. Experimentally it has been found, however that transfused iso-agglutinins, even of a high titer cannot be traced in the circulation of the recipient, even in the course of the transfusion.

In comparing the preservation of plasma in the frozen and the dried states, the following differences are found:-

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In comparing the preservation of plasma in the frozen and the dried states, the following differences are found

1. Preservation of plasma in the dried state effectively prevents bacterial growth and flocculation, but the regenerated material is turbid (as has been said) and has lost almost all of the prothrombin and a portion of the complement. Loss of prothrombin may be largely eliminated by the use of 0.1 per cent solution of citric acid for the regeneration of dried plasma. The Army and Navy require that plasma, whether preserved in dry or frozen state, be filtered before administration

2. Preservation in the frozen state prevents bacterial growth and flocculation and, at the same time, insures almost complete preservation of all specific elements, including prothrombin and complement. After rapid thawing at 37° C., the value is practically equal to that of freshly prepared plasma

The question of preservatives is somewhat controversial. Addition of a preservative to liquid plasma does not completely eliminate the danger of reaction from bacterial contamination, particularly when plasma is separated from citrated blood which has been stored for a number of days. On the other hand, if plasma is properly prepared by a closed method, and has been found sterile and nontoxic by proper bacteriologic studies and animal inoculation, preservation in the frozen state or in the dried form renders the addition of a preservative superfluous. When large doses of plasma are required, the presence of a mercurial preservative may constitute a real danger to the kidneys, especially if the flow of urine is temporarily reduced

The only useful purpose served by the addition of a preservative to plasma is the protection of the material from the moment of the restoration to fluid state, to the moment when it is administered. The best protection during this time is to reduce the interval to a minimum. In any case the amount of preservative should be small (1.35,000 of sodium ethylmercurithiosalicylate [merthiolate] or 1.50,000 of phenylmercuric nitrate). In these concentrations the value of the preservatives is doubtful

Dosage of Plasma—In estimating the dosage of plasma in treatment of shock, it is necessary to consider the extent and degree of the injury as well as the age and physical status of the patient. It is well known that minimal injuries will produce serious systemic reactions if a patient is physically debilitated. In addition, as already has been mentioned, the state of hydration of the patient and the amount of capillary damage must be estimated as well as possible. This is not always easy on the field, since repeated determination of plasma proteins, determination of hemoglobin, estimation by means of the hematocrit, and so forth are not readily done

In the treatment of shock all that is possible must be done to prevent the initiating factor from acting a sufficiently long time to produce clinical manifestations. The best treatment is, in other words, prevention. Patients exposed to obvious and sufficient precipitating factors must be treated as potential sufferers from shock, without waiting for the appearance of clinical symptoms. Thus, a patient who has undergone extensive injury with crushing of tissues and with or without loss of blood, should not be submitted to an extensive operative procedure involving general anesthesia without a dose of 17.5 to 35 gm. of plasma proteins (250 to 500 cc. of undiluted plasma)

Patients who gave manifestations of early moderate shock (cold moist skin, grayish blue color, feeble and rapid pulse, unchanged or slightly lowered pulse pressure) must be treated immediately and adequately, since the management of early shock is, as a rule, a simple and successful procedure, whereas late shock, with severe capillary damage, often is very difficult to combat. This line of demarcation between early shock and late shock, with severe capillary damage, divides the patients who are successfully treated with relatively small doses (17.5 to a maximum of about 52 gm., or 250 to 750 cc. of undiluted plasma) from those to whom larger doses (53 to 105 gm. of plasma proteins, or 750 to 1500 cc. of undiluted plasma and more) must be given repeatedly with only a fair chance of success. These more severe forms, occurring as a rule among patients who, regardless of the severity or nature of the initiating factors, have been allowed to go for a time without adequate treatment, show as typical signs a considerable drop in blood pressure and particularly in pulse pressure, rapid, thready pulse severe reduction of surface temperature, collapsed veins, slow flow of blood from wounds, thirst, hemoconcentration, low urinary output, and occasionally edema. In these cases, maximal doses of plasma must be given (105 to 210 gm. of plasma proteins and up, or 1500 to 3000 cc. of undiluted plasma and more) as a rule the first 250 to 500 cc. must be given in a few minutes. This is accomplished by applying pressure through the air filter with a simple rubber bulb and valve, of the type used in operation of a sphygmomanometer. While it is poor judgment to wait for a drop in blood pressure in order to make a diagnosis of shock, such a drop is a very good index by which to judge of the efficiency and adequacy of treatment of shock in general and of the dosage of plasma in particular.

The value of various criteria of shock already has been discussed. Some have definite value in estimating the effect of therapy in general and of fluid therapy in particular.

It is repeated, to emphasize, that one must not wait for clinical signs of shock in order to institute proper intravenous fluid therapy. Fluid therapy should begin in the presence of conditions that are sufficiently severe to act as initiating factors. These may be multiple fractures, burns, hemorrhage, cold, and so forth. If dehydration is present, solutions of crystalloids should be given along with an initial dose of plasma. Intravenous administration of crystalloids beyond the initial dose is not necessary, even in the presence of dehydration, if the condition of the patient is not very severe and he can take fluids by mouth.

Solutions of crystalloids, however, must not be given, even in the presence of moderate dehydration, if the shock is in a late stage, if it is severe and, particularly, if there is a drop in the total plasma content of the blood. Under these conditions, intravenous addition of solution of crystalloids would tend to dilute the already diluted plasma protein, thus increasing the capillary damage and consequently increasing the loss of plasma in the tissues, particularly the albumins.

After the administration of a quantity of plasma sufficient to restore a proper volume of circulating blood, as judged by pulse, blood pressure, the patient's general condition, and so forth, just enough solution of crystalloids should be administered to maintain a sufficient output of urine, approximately 900 to 1200 cc. An exception should be made in the presence of burns and in other conditions in which a certain degree of renal damage is presumed to have taken place. In these cases the total urinary output should be somewhat higher, in the order of 1500 to 1800 cc., and even higher if possible.

As was said a few paragraphs earlier, while it is poor judgment to wait for a drop in blood pressure in order to make the diagnosis of shock, reappearance of a lowered blood pressure is a very good index by which to judge of the condition of the patient in response to intravenous fluid therapy.

There appears to be no justification, from practical observation, for the *routine* use of plasma proteins in concentrated form. This is true of both whole plasma and purified albumin. It has been noted that the administration of concentrated blood plasma (four to five times concentrated) or of concentrated purified human albumin (18.3 per cent) in sufficiently large doses (50 gm. and more) has a very rapid effect, often producing, in the presence of an open wound, considerable and undesirable bleeding. The only justification for the use of concentrated solutions of plasma, obtained by dissolving plasma dried from the frozen state with less than the original

volume of water, is in the treatment of shock in the presence of head injuries likely to cause edema of the brain.

Purified Albumin of Human Origin

Another blood derivative likely to gain a definite field of usefulness in the treatment of shock under conditions expected to prevail in warfare and similar emergencies, is purified human albumin. The advantage of this material is that, since it is stable in concentrated solution, it can be carried in small containers, ready for use, and administered to patients who are in a state of shock before transportation to places where more adequate treatment with isotonic plasma or whole blood can be instituted.

The use of purified human albumin appears to have a definite limitation in cases of shock attended with widespread capillary damage, particularly in late shock and in burns, in which albumins are more readily lost from the circulation because of their smaller molecular size. Another limitation is the loss of material in the process of preparation.

SOLUTIONS OF CRYSTALLOIDS

While it is true that sodium chloride and glucose solutions do not produce permanent increase in the volume of circulating blood because they readily leave the circulation, their use is of great importance in the treatment of shock.

As has been emphasized elsewhere, in a severe case of shock it is essential to establish venoclysis at the earliest possible moment, even before blood or plasma may be available. The needle is maintained open, with 5 per cent glucose in physiologic saline solution administered by the drip method, at a rate of about 20 to 40 drops per minute.

Another important role of solutions of crystalloids is in the treatment of dehydration, a topic already covered in other portions of this manual. It is generally difficult to relieve dehydration in cases of severe shock by administering water by mouth or rectum, and fluids administered subcutaneously in this condition are not absorbed. The danger of dehydration in cases of shock is generally greater than the danger of administering too large a quantity of fluids parenterally provided they are administered in conjunction with material possessing high osmotic value, such as blood or plasma. The administration of crystalloid solutions alone is definitely contraindicated in cases of late, severe shock when extensive capillary damage has occurred. It may be stated more precisely that solutions of crystalloids never

should be administered to a patient who is in a condition of shock and whose total plasma protein has undergone severe reduction. Hypertonic glucose solution often causes thrombosis of the vein. In late shock it has the same disadvantage as crystalloid solutions, since it draws water into the vascular system. Rapid metabolism of glucose is another drawback to its use.

Because of the frequency with which dehydration is a factor in shock, and because of the important role it plays, especially in injuries of warfare, there appears no justification for the routine employment of hypertonic solution, of either plasma, serum, or purified human albumin.

SOLUTIONS OF ACACIA AND PECTIN

Solution of acacia has been used with some degree of success in the treatment of shock. However, it is stored in the liver, where it causes damage. Its use, therefore, is to be discouraged.

Solution of pectin is likely to prove to be a better substitute for blood than acacia, but as yet it has not been sufficiently tried.

CLINICAL EXAMPLES OF FLUID REPLACEMENT THERAPY IN SHOCK

Five cases of shock illustrating various points of dosage are here reported. Cases 1 and 2 illustrate severe, early shock, readily and permanently relieved by relatively small amounts of plasma. Case 3 emphasizes the losses of blood plasma in a case of shock involving extensive capillary damage. Case 4 is an instance of severe shock in which an inadequate amount of plasma was given with fatal result. Case 5 is one of severe shock, in many ways similar to Case 4; treatment was with large amounts of plasma and recovery ensued despite extreme loss of blood and old age.

Case 1.—A white man, thirty-one years of age, was admitted to the accident ward of a hospital at 12:30 A. M. The patient was suffering from a compound fracture of, and dislocation of, the acromion and clavicle, compound fracture of the left humerus, and complete avulsion of the deltoid muscle. A car, driven by the patient, had been hit at a road crossing, the door had swung open, and the patient was thrown out violently, striking the curb with the left shoulder. At 2:15 P. M. the patient's blood pressures, in millimeters of mercury, were 138 systolic and 92 diastolic. At 3 P. M. he was sent to the operating room. The operation for débridement, with wiring of the shoulder, lasted two hours and twelve minutes. Ether anesthesia was em-

played. About one hour after the beginning of the operation, the patient went into deep shock, with a pulse rate of 140 per minute. The pulse also was thready and very weak and the blood pressures were 62 and 26. At this time the shock was so severe that the operation was interrupted and all efforts were directed to combat shock. Plasma in a quantity of 750 cc. was administered. The patient recovered from shock in about forty-five minutes and the operation was successfully completed. On return to his room at 5 45 P.M., the blood pressures were 128 and 60. The patient made an uneventful recovery.

Case 2.—A white man, thirty-nine years of age, was admitted to hospital at 1 30 P.M. The patient was suffering with compound fracture of the left tibia, fibula, and left humerus, and laceration of the scalp. Loss of blood was not excessive. The patient had jumped, with suicidal intent, in front of a moving automobile about half an hour before admission. A partial record of subsequent events is given in Table 1.

TABLE 1.—PATIENT'S RECORD

Time, P.M.	Pulse Rate per Minute	Blood Pressure, Mm. of Mercury		General Conditions	Intravenous Fluid Therapy
		Systolic	Diastolic		
1:45	70	84	56	Clinically in shock	5 per cent glucose in physiologic NaCl im- mediately followed by plasma (17.5 gm. of plasma proteins or 250 cc. undiluted plasma)
3:30	90	80	60	Clinically in shock	
4:00	90	110	80	?	
4:45	90	94	54		17.5 gm. plasma pro- teins (250 cc. undilu- ted plasma)
5:15	80	130	80	Improved enough to warrant op- eration	
6:10-8				In operating room	

During the operation the blood pressures varied from highs of 110 and 60 to lows of 60 and 40. The patient received a third dose of 17.5 gm. of plasma proteins. He left the operating room clinically in good condition. At 10 P.M. his pulse rate was 84, his blood pressures, 124 and 78; he gave no evidence of shock.

The patient made an uneventful recovery.

Case 3.—A white woman, seventy-two years of age, was admitted to hospital at 10:45 A.M. She had received the diagnosis of pelvic tumor, based on symptoms of lower abdominal pain, pressure over the bladder and frequency of urination. Also palpation had disclosed a hard mass in the lower, middle portion of the abdomen, extending above the pubis to a point just below the umbilicus. On the afternoon following admission, the patient complained of abdominal discomfort. The abdomen was slightly distended. Examination gave to the attending physician the impression that the pelvic tumor was smaller than it had been, and this was attributed to the distended intestine. On admission, the systolic blood pressure was 190 mm. of mercury and the diastolic, 70, normal for the patient. At 3 P.M., the blood pressures had dropped to 118 and 80. At 7 P.M., the nurse noted that the patient was apparently comfortable and at 8 P.M., the pulse rate was 80 per minute. From 10 P.M. to 2 A.M. of the following day, the nurse noted that the patient was apparently sleeping; at 3 A.M., the patient was found dead and cold.

Necropsy, performed approximately six hours after death, disclosed about 2000 cc. of turbid, brownish fluid in the abdominal cavity, which apparently had escaped from a large, ruptured, papilliferous, serous cystadenoma of the ovary. The entire peritoneal surface was congested, the intestinal wall was edematous, and the ileum contained more than 2000 cc. of turbid fluid, the total protein content of which was 1.5 per cent (micro-Kjeldahl). The albumin content of this fluid was 1.3 per cent; globulin, 0.2 per cent. There was, in addition, edema of the lungs. In the heart and other organs pathologic lesions were not found. Thus, the total loss of proteins in the lumen of the ileum alone was 30 gm., of which 26 gm. were albumin. This is the hydrostatic equivalent of more than 600 cc. of plasma-albumins, or more than 1 liter of whole blood. If it is considered that the wall of the intestine, the colon, the loose tissues of the parietal peritoneum, and the lungs contained comparatively large quantities of transudate, the magnitude of the loss becomes apparent.

The rupture of the ovarian cyst in Case 3 apparently occurred sometime after the patient's admission to the hospital, probably before 3 P.M. Death was clearly attributable to shock from chemical irritation of the peritoneal surface by the escaped content of the ovarian cyst. This case illustrates the extreme speed with which loss of protein, especially albumin, occurs when generalized capillary damage is present. This picture is similar to that seen in late shock, regardless of the nature and location of the initiating factor.

Case 4—A white man, seventy-five years of age, was admitted to hospital at 6 30 P.M. The patient was suffering from a compound fracture of the right tibia and fibula and other undiagnosed injuries, but loss of blood was negligible. The patient was struck by a car shortly before admission. In the accident ward his blood pressures varied from 90 mm. of mercury systolic and 50 diastolic to 110 systolic and 80 diastolic. On arrival in the operating room at 7 40 P.M., the blood pressures were 86 and 45 and the pulse rate 110 per minute. The patient was clinically considered to be in a state of shock and to constitute a poor operative risk.

The operation was begun at 8 13 P.M. and consisted of débridement of the compound fracture of the tibia and fibula, packing with vaseline gauze and application of a plaster cast. For these procedures the patient was under nitrous oxide and oxygen anaesthesia. At 8 15 P.M., the blood pressures had dropped to 60 and 40 and the pulse rate was 120. Transfusion of plasma was begun with 450 cc., administered during the operative procedure. On the patient's leaving the operating room at 9 P.M., blood pressures were 85 and 60 and the pulse rate 130, the pulse, also, was thready and irregular. On return to his room, at 9 15 P.M., the patient was thirsty and restless and complained of pains in the back. At 10 20 P.M. the pulse rate was 108, blood pressures were 80 and 50 and the patient was restless. At 11 20 P.M. the pulse rate was 114, blood pressures were 56 and 30 and the skin was cold. At 1 A.M. of the following day, systolic blood pressure was 54 and the diastolic pressure was questionable the pulse was very weak and its rate was about 120, respirations were 40 per minute and were shallow. The patient was cold and complained of constant pain. At 1 45 P.M. Cheyne-Stokes respirations set in, pulse and blood pressures became imperceptible, and the patient died at 2 A.M. Fluid was not administered intravenously after the operation.

Necropsy revealed, in addition to the fracture of the leg, fracture of the pelvis, fracture of the fifth thoracic vertebra without displacement, and fracture of ten ribs on the left side. The intestinal wall was edematous and the lungs congested and edematous.

Case 5—A white woman, seventy three years of age, moderately obese, had been riding in a car with four other occupants when the car hit a tree. The patient presented the following injuries: compound fracture and dislocation of the right elbow, compound fracture and dislocation of the left ankle, compound fracture of the right tibia and ankle, fracture of six ribs on the left side with compression and profuse hemorrhage. The sequence of events, best given in outline form, is as follows:

12 noon	Accident.
1:15 P M	Admitted to accident ward of hospital, patient regained consciousness on admission
1 35 P M	Radial pulse not obtainable; heart beats 80 per minute, blood pressures 62 mm of mercury systolic and 46 diastolic; clinically, patient in a state of severe shock.
1 40 P M.	60 cc. of 183 per cent purified human albumin solution given (11 gm of albumin) in about two minutes
1-50 P.M.	Radial pulse became perceptible, blood pressures 88 and 52 Transfusion of plasma begun at 1 50 P.M. and, by 2 15 P M., 250 cc had been given.
2 15 P M	Pulse rate 80, pulse better quality, blood pressures 90 and 60 Administration of the second lot of plasma was begun
2 30 P M	250 cc of undiluted plasma administered (or 500 cc total in forty minutes)
2-40 P M	Blood pressures 86 and 54, pulse rate 86. Patient appeared much improved. Administration of 500 cc of 5 per cent glucose in physiologic saline solution was begun and was completed at 3:55 P M.
3-55 P M.	Blood pressures 80 and 40, pulse rate 80 Morphine $\frac{1}{4}$ grain (0.016 gm) administered subcutaneously
4-30 P M	Patient's skin cold, moist, axillary temperature 97.6° F, blood pressures 62 and 40; pulse rate 80. Administration of 500 cc of undiluted plasma started and was completed at 5 30 P.M. (or 500 cc. in one hour)
5-45 P.M.	Blood pressures 80 and 56; pulse rate 96; patient generally improved over condition recorded at 4.30 P.M. Administration of 5 per cent glucose in physiologic saline solution was begun and 500 cc were administered by 8:30 P.M.
6:10 P.M	Blood pressures 94 and 54, pulse rate 94 Patient generally improved
6:45 P M.	Blood pressures 110 and 58, pulse rate 96. Patient's improvement continued.
7:40 P M	Blood pressures 116 and 66; pulse rate 86.
8:30 P M.	Blood pressures 110 and 62, pulse rate 100. At 8-40 P.M. intravenous administration of 500 cc. of glucose 5 per cent in physiologic saline solution started and was completed at 11-45 P M.

- 9 20 P.M. Blood pressures 110 and 60 pulse rate 96. Kirschner wire inserted in right ankle. Oozing of blood from right leg continued.
- 11 00 P.M. Blood pressures 116 and 62 pulse rate 96 axillary temperature 99.2° F
- 11 45 P.M. Administration of 300 cc. of glucose 5 per cent in physiologic saline solution started and was completed at 1 30 A.M.
- 12 midnight Blood pressures 106 and 60 pulse rate 100 Patient restless.
- 1 20 A.M. Blood pressures 92 and 52 pulse rate 100. Patient restless general condition appeared worse.
- 1 30 A.M. Administration of 500 cc. of plasma started and, at the same time, oxygen given intranasally. The plasma was run slowly and the administration was completed at 6 25 A.M., or in four hours and fifty five minutes.
- 2 00 A.M. Blood pressures 90 and 60 pulse rate 100
- 2 40 A.M. Blood pressures 100 and 58 pulse rate 108 temperature 100.3° F
- 4 00 A.M. Blood pressures 100 and 58 pulse rate 98 temperature 100.3° F Patient appeared generally better
- 5 00 A.M. Blood pressures 100 and 60 pulse rate 104
- 6 25 A.M. Blood pressures 100 and 60 pulse rate 100 temperature 100.3° F Total undiluted plasma administered 1500 cc. or approximately 105 gm. of plasma proteins.
- 7 00 A.M. Blood pressures 136 and 92 pulse rate 96 temperature 100.4° F
- 10 27 A.M. There were 5.1 gm. of hemoglobin per 100 cc. of blood and red blood cells numbered 1,900,000 per cubic millimeter of blood. Transfusion of 500 cc. of whole blood was begun, and, at 3 05 P.M. on the afternoon of the following day 500 cc. more was given.

The patient recovered.

The variations in blood pressure, pulse rate, concentration of hemoglobin, and in total protein, albumin, and globulin content of the blood are graphically represented in Fig. 5

An analysis of the graph of Case 5 shows that the administration of 500 cc. of 5 per cent glucose in physiologic saline solution between

2 40 and 3 55 P M was followed by hemoconcentration, with a sharp rise of the hemoglobin content from 5.5 to 8.1 gm in a period of about an hour. At the same time there was a drop in the blood pressures from 86 and 54 to 62 and 40 and a drop in the total protein content. The condition of the patient at this time was definitely worse

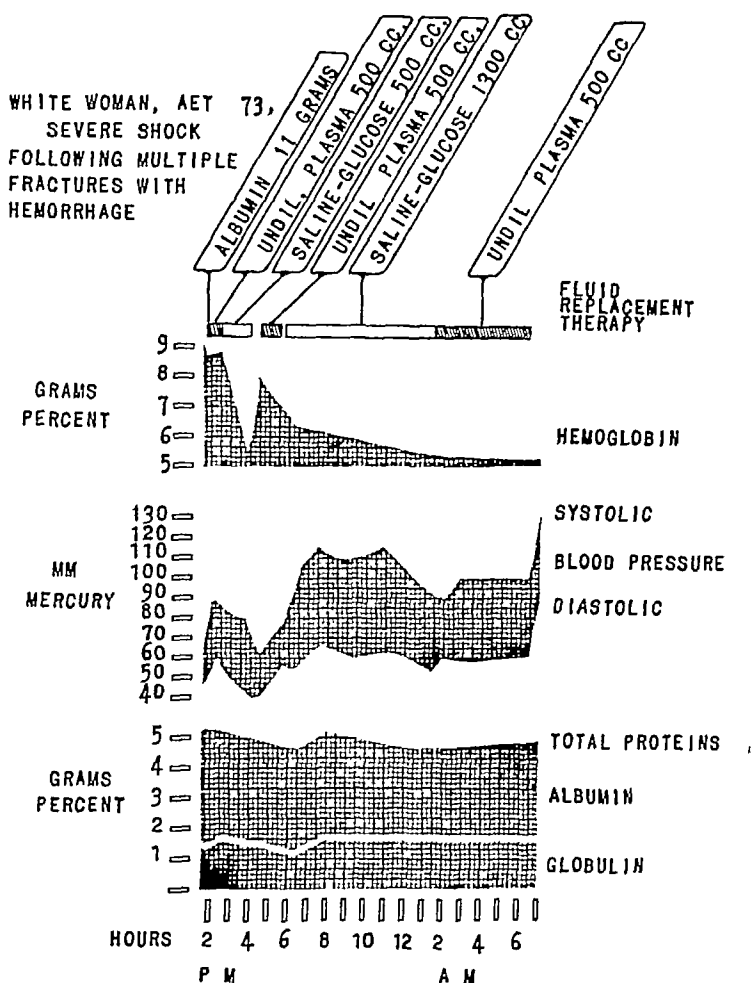


Fig 5—Data on Case 5

In view of the fact that this patient presented, at the time, moderate but definite hypoproteinemia, intravenous administration of the glucose in saline solution should have been avoided. Later on, when the patient had recovered fairly well from shock following the administration of an additional 500 cc. of undiluted plasma, administration of glucose in saline solution (1300 cc from 5 45 P M. to 1 30 A M) was tolerated much better. However, another drop in the blood pressure occurred at 1 20 A M., with deterioration in the general condition of the patient. The condition was relieved by the last administration of plasma (500 cc. from 1 30 A M to 6 25 A M)

This illustrates the danger of administration of crystalloid solution in a severe case of shock, and the necessity of constant and close supervision in such cases because, as indicated by the course of Case 4 a fatal outcome may occur very rapidly unless treatment is carried out properly and for a sufficiently long time.

BURNS*

Fluid therapy for burns is, in the early phase, more or less similar to that of shock, later on, however continued hypoproteinemia, in part resulting from losses of plasma in damaged tissues, constitutes a trying problem for the maintenance of proper electrolyte and fluid balance.

It must be kept in mind that shock may occur in association with comparatively superficial burns, if they are sufficiently extensive. The mild to moderate burn, however with little or no systemic reaction, usually is treated readily and, as a rule, intravenous administration of fluid is needed only for the first day or a little more. The following management of burns, so far as the intravenous fluid therapy is concerned, is intended for moderate to severe cases of burn, with burned areas of varying extent, usually associated with shock in the first hours and later on with hypoproteinemia, edema, electrolyte imbalance and so on.

As quickly as possible, venipuncture is performed to obtain a specimen of blood, the needle or cannula is left in place, and administration of fluids is begun at once. If plasma is not available for use at once, as it may be necessary to restore it to fluid form from the frozen or dried states, the needle or cannula is maintained open by the continuous drip method, employing minimal amounts of 5 per cent glucose in physiologic saline solution (about 20 drops, or 1 cc, per minute). Hematologic and chemical studies are carried out immediately if possible, on the specimens of blood collected. In order of importance they are the estimation of hemoglobin, the total protein content (by the falling-drop method) chlorides and carbon-dioxide combining power of plasma. The determinations must be repeated every four or six hours for the first twenty four hours for the next nine to fourteen days, every twenty four hours is usually sufficient, according to the progress of the patient's condition.

Red-cell counts, white-cell counts, studies of the leukocytic formula, and determination of urea are useful but not necessary. Estimation by hematocrit may be substituted for the determination of hemoglobin if the latter cannot be made with a method insuring an exact

* See also the manual on burns, with which this volume opens.

reading, such as that obtained with a well-calibrated photo-electric colorimeter

The main problem in the early stages is, of course, generally one of shock, usually complicated by severe hemoconcentration. Immediately following the burn, frequently all the formed elements are increased, the patient is dehydrated in the sense that the fluid no longer is in the vascular bed but in the damaged tissues. The hemoconcentration with increased viscosity, and the diminished volume of circulating blood, result in progressive peripheral anoxia, widespread capillary damage and further loss of plasma and fluid, particularly in the splanchnic region and in the lungs. At this point fluids are required. At the same time, or very shortly thereafter, it is noted that although the formed elements of the blood are usually still concentrated, hypoproteinemia has begun to develop. It is at this point that administration of crystalloid solutions and of whole blood is contraindicated but plasma is needed in large quantities. The following case illustrates this point.

Case 6.—A white woman, thirty-five years of age, was admitted to hospital at 11 45 A.M. January 19, 1939, suffering from second and third degree burns of both legs, thighs, buttocks, arms, and hands, the result of ignition of a negligee made of inflammable synthetic silk. Determinations made two and one-half hours after the accident disclosed severe hemoconcentration, not accompanied by hypoproteinemia but by alteration of the albumin-globulin ratio, as shown by the following figures: hemoglobin 19.5 gm per 100 cc. of blood, red blood cells 6,700,000 and white blood cells 16,500 per cubic millimeter of blood, total serum proteins 7.9 gm, albumin 3.8 gm., and globulin 4.1 gm., per 100 cc. By 9 A.M. of January 20, or nearly twenty-four hours after the burns had been inflicted, the patient had received 3250 cc of 5 per cent glucose in physiologic saline solution, 3250 cc. of 5 per cent glucose in water, as well as 650 cc. of blood plasma, or a total of 7150 cc of fluids. At this time, the concentration of hemoglobin was 19.2 gm per 100 cc and red blood cells numbered 6,640,000 per cubic millimeter of blood. The value for total serum proteins was 6.9 gm per 100 cc. On January 21, forty-eight hours after the burns had been sustained, the total serum proteins were 6.2 gm. per 100 cc. despite the additional administration of 300 cc. of plasma.

Plasma may be fresh, or stored in the frozen or dried state. There is obviously no need for the use of hypertonic material.

In the days following the period of shock, long-sustained anoxia and hypoproteinemia, often associated with toxemia of infection, produce marked changes in, and dysfunction of, kidneys, liver and other vital organs. Anemia usually develops, often with increase in concentration of serum bilirubin hypoproteinemia, and particularly hypo-albuminemia, persist. The problem of edema is particularly serious. This condition is due, to a great extent, to hypoproteinemia, although, in some cases, prolonged and excessive hydration with crystalloid solutions and renal damage may play parts. Hypoproteinemia must be treated with large doses of plasma, alternated after the second or third day with transfusion of whole blood for replacement of lost erythrocytes, in proportion to the loss. The loss of plasma proteins through the areas denuded of epithelium may be reduced by tanning, which should be carried out as early as possible. However the losses of plasma in the damaged tissues cannot be avoided and the lost material must be replaced. The quantity of plasma necessary for the treatment of a severely burned patient is usually very large. For the treatment of shock in the first twenty four hours, the amount required is usually from 52.5 to 72 gm. of plasma proteins (750 to 1000 cc. of undiluted plasma). The requirement for the next three or four days is only slightly smaller (35 to 52.5 gm. of plasma proteins or 500 to 750 cc. of undiluted plasma). Thereafter the required amount of plasma varies a great deal from case to case, depending on the extent and depth of the burn, infection, renal damage, weight of the patient, and so forth. The following examples give an idea of the total dosage

Case 7.—A white woman, seventeen years of age, sustained first and second degree burns of the face, neck, entire thorax, both arms, and both hands, 60 per cent of the body surface was affected. She required 383 gm. of plasma proteins in eleven days (5400 cc. of undiluted plasma). The patient made a rapid and uneventful recovery

Case 8.—A white woman of twenty-seven years had second and third degree burns of the right hand, third degree burns of the entire legs and of the dorsums of both feet. She required 175 gm. of plasma proteins in seven days (2500 cc. of undiluted plasma). The patient made a rapid and uneventful recovery

Case 9.—A white man, thirty two years of age, had second and third degree burns of the face, neck, hands, and entire surface of the legs. He required 407.5 gm. of plasma protein in fourteen days

(7350 cc. of undiluted plasma), the osmotic equivalent of nearly twenty-nine transfusions of 500 cc. of whole blood. The patient died suddenly, on the fifteenth day, of duodenal hemorrhage.

Case 10.—A white woman, twenty-seven years of age, sustained second and third degree burns of the face, neck, both forearms and hands, and both legs, from above the knee downward, including the dorsums of both feet. She required 175 gm. of plasma proteins (2500 cc. of undiluted plasma) in five days. The patient had made a good recovery from the burns and was in the hospital for skin grafting of the burned areas when she died suddenly of pulmonary embolism, three and one-half months after the accident.

It is to be noted that in Cases 7 to 10 inclusive, the shock was rapidly brought under control and that there was freedom from edema although the burns were severe and extensive in all cases. At necropsy, in Case 9, the liver and kidneys were found in good condition. This and similar cases suggest that the so-called toxic changes of the liver and kidneys probably are due to sustained hypoproteinemia.

The intake of fluid of patients suffering from burns usually is very large in the first twenty-four hours, averaging from 6000 to 7000 cc. Patients with burns not infrequently vomit and for this reason it is often necessary to give fluids by routes other than the oral one, usually intravenously. The quantity given must be large enough, in any case, to obtain a relatively large amount of urine, approximately twice the average normal, or about 2400 cc daily, in view of the likelihood of renal damage and of the necessity of a larger output to obtain satisfactory elimination of solutes. The importance of prompt and proper tanning in limiting the loss of plasma protein in cases of burn is readily understood. In one case, that of a young woman, it was necessary to remove the tanned surface because of streptococcic infection and formation of pus under the eschar. In one period of twenty-four hours, the losses of plasma protein from one forearm alone were 115 gm, or the equivalent of approximately 164 cc. of undiluted plasma. In all cases of severe burns, teamwork between laboratory and clinician, so important in all phases of proper practice of medicine, is essential.

POSTHEMORRHAGIC SHOCK

In the fluid therapy of posthemorrhagic shock, distinction must be made between the cases in which the bleeding has ceased and the

WOUND HEALING

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WOUND HEALING

CHAPTER I

FACTORS IN HEALING AND SEQUENCE OF EVENTS

HEALING of wounds is a composite biologic phenomenon which conforms, in general, to the laws of growth, having a *latent* or *lag period* and an *active period* of cell growth. The processes involved interact with one another but there are quantitative variations of each, depending on the tissue involved in relation to the condition present in the wound. The time required to establish a balance between the dead and the living cells and to eliminate necrotic material and foreign bodies—including bacteria—determines the length of the lag period. The three most important processes involved in the second period of cell growth or fibroplasia are (1) ameboid movement, (2) mitotic proliferation, (3) maturation of cells engaged in fibroplasia and fusion of the wound surfaces.

LOCAL FACTORS

The lag period and the period of fibroplasia, that is, the healing of the wound, are determined by the following local factors (1) The amount of necrotic and damaged tissue in, and bordering on, the wound is related to healing. (2) The vascularity of the tissues involved is another element. The more abundant the capillary bed, the more rapid is the repair. The integrity of the blood supply determines the nutrition and the viability of the tissues bordering on the surfaces of the wound. (3) Still another set of factors are the amount and the character of the exudate in the wound space and in the bordering tissues. (4) The number and virulence of infecting bacteria in the wound spaces and in the bordering tissues also are important features. (5) The final factor embraces the number and character of foreign bodies in the wound, to be extruded or encapsulated.

SYSTEMIC CONDITIONS

Of these, the conditions which determine the duration of the lag period and which have a profound influence on one or more of the local factors are the following (1) The age of the tissues is important; that is, whether they are adolescent, normal adult or senes-

cent, or degenerated (2) The state of normal hydration also has an effect. Dehydration or overhydration of the tissues is determined by the water, electrolyte and protein balance in the blood and an imbalance may profoundly alter the conditions in the contents of the wound space and in the bordering tissues. (3) Another determining systemic condition is the normal nutritional balance. Protein deficiency retards, high protein diet accelerates, wound healing A fat diet prolongs repair of wounds (4) Still another factor is the vitamin balance C avitaminosis and C vitamin deficiency definitely prolong the lag period because this vitamin plays an essential role in the formation of intercellular substance, in maturation of the fibroblast and in transformation of fibrous tissue to collagen fibers (5) The final conditions which must be taken into account are the state of the general circulation and the blood picture. Poor circulation and severe anemia definitely alter or delay wound healing These systemic conditions will come up for consideration again before this chapter closes

SEQUENCE OF SIMPLE HEALING

In the study of wound healing, the least complicated sequence of the reparative processes is seen in the cleanly incised wound, made and closed with the strictest aseptic, hemostatic, and atraumatic technic. For this reason, such a wound is of the greatest interest to both biologist and surgeon In such a wound the lag period of four days is uniform provided none of the systemic deleterious factors are present. The initial escape of blood and plasma is minimal, the formation of a mesh of fibrin from the plasma is not delayed by the necessity of eliminating dead tissue by autolysis and phagocytosis, and the excessive exudate of the bacterial and foreign body reaction does not widely separate the surfaces of the wound In such a wound, the quiescent period of agglutination of surfaces by a thin layer of fibrin is short.

AMEBOID MOVEMENT AND PROLIFERATION OF CONNECTIVE-TISSUE CELLS

There would seem to be some definite factor initiating the ameboid movement of new connective-tissue cells, but just what this X factor is has not been demonstrated as yet. This growth stimulating factor or substance is probably derived from damaged cells in the surfaces of the wound. In tissue culture, crushed embryonic tissue juices definitely stimulate cell growth. Another theory is that the inadequacy of the supply of oxygen and of other nutritive substances

in the ischemic surfaces of the wound leads to a tissue hunger which initiates ameboid movement and cell division.

The destructive or lytic phase of the lag period, by which dead tissue is removed by autolysis and phagocytosis, is succeeded by ameboid movement, into the firmous zone, of fibroblasts, derived not from the adjacent fixed connective tissues, but from the wandering connective tissue cells, fibroblasts and histiocytes. At this period mitotic proliferation of these mesenchymal cells is accelerated.

During the lag period two processes connected with the ameboid movement and proliferation of fibroblasts would seem to take place. The first is the phenomenon of stereotropic response of growing cells to surfaces. Fibroblasts in contact with strands of fibrin or fibrils have a strong tendency to elongate and to grow along the fibrils, just as epithelial cells manifest ameboid movement along plane surfaces in tissue culture, or along granulation tissue or beneath a scab. The second process is the centrifugal force which directs, or pushes, the cells away from their own tissue into the mass of plasma in the wound space. This process induces various cells, including fibroblasts, to move into blood clot in a fanlike manner, to take part in organization of the clot. Similarly endothelial buds give evidence of centrifugal growth into organizing fibrin, with consequent spread of the vascular bed and the formation of granulation tissue.

With maturation of the fibroblasts and their elongation along fibrils of fibrin, uniting the wound surfaces, and with the development of collagen fibers from the elongated fibroblasts, there takes place the change from the lag period, with no appreciable tensile strength in the wound, to the second phase of wound healing, the period of fibroplasia, characterized by a sudden and rapid increase in tensile strength.

HOW SURGICAL METHODS AID OR HINDER HEALING

From the standpoint of practical surgery, the lag period is the interval between the receipt of the wound and the beginning of tensile strength, during which time the surfaces of the wound have to be held together by mechanical means, by sutures, by splinting or by constantly maintained pressure. These measures of maintaining apposition must be carried out with the least damage to the wound surfaces and bordering tissues, with maintenance of maximal nutrition, adequate blood supply, minimal foreign body reaction and maintenance of rest by immobilization. This is the period of repair of the wound, when the surgeon can contribute the most by the intelligent use of his art.

It is in his efforts to insure and maintain apposition that the thoughtless surgeon makes his most common mistake of suturing the edges of the wound and the individual layers too tightly—and with suture material out of all proportion to the holding strength of the tissues. Undue tension in the sutures will cause wide zones of anemic, even ischemic, tissue, thus prolonging the lag period by increasing the lytic process in the wound.

In no field of surgery is this factor of tension ischemia better illustrated than in intestinal anastomosis. If the tension on the suture line is increased by distention resulting from intestinal gas, necrosis will occur in the suture line, with resulting leakage. Whereas if the bowel can be kept deflated with the use of intestinal intubation, healing takes place rapidly and uneventfully.

Systemic Conditions Again

It is in the understanding of the systemic factors of healing of wounds, mentioned early in this chapter, that the most recent advances have been made. In this field the science of surgery adds immeasurably to the art of wound repair. Attention is called to these factors outside of the wound in the management of the wounded individual.

Influence of Age—Clinically it always has been known that wounds heal more rapidly and satisfactorily if the patient is young than if he is old. It becomes the more important to maintain nutrition of tissue and to avoid the local deleterious factors of infection and foreign body reaction in repairing wounds of the old patient and in senescent, poorly nourished tissue.

Normal Fluid, Electrolyte, and Protein Balance.—Extreme degrees of dehydration, as seen in prolonged or severe loss of fluids and electrolytes, deplete intercellular fluid and may disturb the normal intercellular salt and fluid balance, which will threaten not only wound healing but the patient himself. Overhydration may cause as serious disturbance in wound healing and prolongation of the lag period as dehydration, for edema of the tissues definitely delays the onset and the progress of fibroplasia.

Methods now perfected for determining fluid and salt balance, that is: (1) the hematocrit for determining concentration of blood, and (2) the falling-drop method for determining the specific gravity of the blood and plasma and the percentage of protein in the blood, as well as (3) acid-base determinations, are essential in following the course of patients operated on or severely injured. These methods give definite information as to the needs for fluid and electrolytes and prevent overdosage.

Deficiency of protein in the blood may be caused by prolonged starvation with respect to proteins, or by loss of protein following severe or repeated hemorrhage, continued inflammatory exudate or drainage from a fistula. This hypoproteinemia, because of the reduced concentration in the blood of substances of large molecular structure, results in loss of fluid from the capillary bed into the intercellular spaces and in tissue edema. In the presence of such deficiency of protein, edges of wounds appear soggy with edema and give no evidence of fibroplasia as late as the eighth to the fourteenth day. This results frequently in disruption and evisceration following abdominal wounds, if the patients are chronically ill and cachectic. In such cases, it is essential to determine the values for protein in the blood and to combat hypoproteinemia by means of transfusions of plasma and a diet high in protein before and after operation.

Normal Nutritional Balance.—Maintenance of values for protein, aside from its effect on intercellular fluid, is necessary to provide cellular nutrition. The manner in which tissues obtain their nitrogen and build up their new protoplasm still remains a mystery, but the fact is that tissues in the wound require protein as do body tissues elsewhere.

A diet high in protein influences the total period of healing as well as shortening the lag period.

Vitamin Balance.—From the enormous amount of research undertaken to establish the role of the many vitamins in tissue metabolism, two vitamins have been shown to have special significance in relation to healing of wounds. These are vitamin C and vitamin K.

With reference to *vitamin C* it is now well established that intercellular substance in general, and especially in the capillary bed, and that the collagen of all fibrous tissue, require ascorbic acid for their production and maintenance. Lack of, or deficiency of, intercellular substance in the capillary bed results in hemorrhage into the wound space and in the bordering tissue, prolonging the lag period and delaying fibroplasia and tensile strength in the wound.

Methods of determining the concentration of vitamin C in the blood are still inaccurate but are being refined. There is still some uncertainty as to the length of time the tissues of man can remain depleted of vitamin C, before the man will show signs of scurvy but the role of vitamin C in the formation of intercellular cement substance and collagen seems now definitely established. Deficiency of vitamin C should be suspected if any patient has been on a restricted diet or an inadequate diet, deficient in fruit and vegetables.

The more recently studied *vitamin K* has an essential role in the control of hemorrhage in relation to deficiency of prothrombin, and

it is of great importance if patients are jaundiced or if, because of a biliary fistula, bile is not entering the intestinal tract. Wounds of such patients, or of patients with badly damaged livers, will not heal because of continued bleeding. The administration of vitamin K with bile salts is essential for several days before and after operation to insure clotting of blood and to prevent continuous oozing from the wound.

Circulatory Imbalance and Anemia.—With present-day methods for determining cardiac output and myocardial efficiency, cardiovascular competence can be definitely determined before operation or in examination of the injured individual. With the amazing development of blood banks and the registering of blood donors, and with the facilities that exist for transfusion of blood substitutes, there is no excuse for operating on an anemic individual. Here again counts of blood cells, determinations by means of the hematocrit and determinations of blood plasma have added immeasurably to the scientific care of the anemic and depleted individual before, during and after operation.

CHAPTER II

TRAUMATIC WOUNDS

THE words "wound" and "trauma" are synonymous, but the term "traumatic wound" is understood to mean a wound caused by mechanical violence. Such a wound is always contaminated with bacteria and is potentially infected—if not properly treated within six to twelve hours.

TYPES OF WOUNDS AND OF MISSILES

Modern mechanized life has enormously increased the number and severity of compound, crushing and penetrating, lacerated wounds. Factories and foundries, motorized traffic on land and sea and in the air in civil life and in war and modern methods of warfare, are accountable for these wounds and have made their treatment one of the most serious and important duties of the surgeon. These wounds differ however from the simple incised wound previously described only in the extent of the damage to the tissues and in the degree of associated bacterial contamination and foreign body content. The processes of repair and of reaction to injury and inflammation are merely more pronounced and the period of healing is more prolonged than they are in the case of simple incised wounds. Treatment of these severe wounds, whether carried out in civil or in military hospitals, is based on the same biologic principles of wound healing that previously have been discussed. The results of surgical care of these serious wounds always will depend on (1) the surgeon's understanding of these principles in relation to the variations in the extent of the trauma and to the contamination or infection accompanying such wounds, and (2) his ability to follow these principles as that ability is affected by the conditions met with when he first sees the injured individual and by the availability or lack, of facilities for carrying out the proper treatment, both immediate and late.

Modern warfare, as demonstrated in the recent Spanish Civil War and the present world conflict, causes widespread injury to the civilian population, to men, women, and children, as well as to the military and naval units engaged. Air bombing and high-explosive shellfire have resulted in an enormous increase in the number of crushing in

juries and compound lacerated wounds, caused by shell fragments of low velocity. On the other hand, there has been a great reduction in the number of penetrating wounds from missiles of high velocity and in simple incised wounds. In the Franco-Prussian War of 1870, 90 per cent of the wounds were caused by bullets and less than 9 per cent by shellfire. Modern war results in approximately 80 per cent of wounds caused by projectiles or fragments of low velocity, due to shellfire and bombing.

INFECTION IN TRAUMATIC WOUNDS

The crushing injuries and the lacerated, contused, penetrating wounds are always contaminated by soiled skin and clothing and by superficially or deeply placed foreign bodies. Levaditi demonstrated this in bacteriologic study of 317 fresh war wounds. Cultures from 93 per cent were positive, from 32 per cent three species of organisms were recovered, from 19 per cent, four species; from 23 per cent, five or six species, and from 19 per cent, one species. Cultures from only 7 per cent were negative.

Infection in traumatic wounds, civil or military, progresses along fairly definite lines, in two stages. In the *first stage*—depending on the degree of devitalization, on the quantity and type of foreign body, and on the number and virulence of the organisms introduced into the wound—bacteria remain more or less inactive on the superficial or deep surfaces of the wound for a latent period of four to twelve hours. This is the stage of contamination, and is the period during which the devitalized tissues and foreign bodies in the wound can be excised by débridement. It cannot be too strongly emphasized that healthy, living cells lining a clean wound, or one which has been subjected to débridement, have an amazing capacity for inhibiting bacterial growth, whereas damaged and devitalized tissues in a wound not only do not prevent bacterial growth but favor it.

It may well be asked how one identifies devitalized tissue. This depends on the tissue and the type of wound. If the injured individual has received a deeply penetrating wound, due to shrapnel or a shell fragment of low velocity, or to impact of some other missile, the wound will have to be enlarged to determine the extent of damage to tissues. If the wound is due to a bullet of high velocity, if bone is not shattered and if the projectile has passed through the tissues, the damage to be looked for is in vessels and nerves. If these are not injured, the wound may be treated expectantly. If a penetrating, lacerated, contused wound is present, the wound should be thoroughly cleansed and then examined after being enlarged. Foreign

bodies, loose fragments of bone and lacerated tissue devoid of blood supply should be excised. Contused muscle which has lost the property of contractility, and muscle that does not bleed, should be excised. Skin that is obviously necrotic should be removed, but thoroughly cleansed skin flaps that appear pale but that are connected to healthy skin may be left in contact with underlying vascular tissue. Of course the viability of damaged tissue depends on the integrity of the main blood vessels which supply the part injured. If these are severed or injured, the possibility of saving the tissue, or of treating it successfully by débridement, is greatly reduced and such wounds have to be watched most carefully to determine whether necrosis is increasing.

In the *second stage* the stage of spreading infection, bacteria multiply rapidly in the media provided by necrotic tissue, the blood clot and the extravasated lymph and serum in the wound space, and the bacteria invade the bordering tissues and frequently the blood stream. This results in sepsis in the wound, with constitutional reaction outside of the wound itself. In this stage of infection, débridement is indicated to remove necrotic tissue and foreign bodies but the wound must not be closed. It must be left open and treated by drainage or packing; secondary healing with granulation tissue must be awaited or delayed closure made. Rest of the wound by immobilization, and of the patient by sedation, is even more important than when a contaminated wound has been subjected to débridement. It must be emphasized that granulation tissue is necessary to fill in the wounded surfaces and to protect them from secondary bacterial invasion and infection.

NEW KNOWLEDGE

During the past five years new developments in mechanized warfare, new experience derived from the recent struggle in Spain and from the current wars in Europe and Asia (also the development of drugs of the sulfonamide group, which inhibit the growth of bacteria, and of compounds that liberate oxygen for combating anaerobic bacteria) have added to knowledge of the treatment of war wounds that had been obtained during the war of 1914-1918.

Prevention of Penetrating Lacerated Wounds

Certain advances have been made in this respect, notably introduction of the steel helmet. In the French army the incidence of cranial wounds was reduced from 15 to 3 per cent by its use. Armor made of compressed fiber or plastic material is being tried in the

German army as a protection against thoracic and abdominal wounds. The English are studying the entire problem of the "physical prophylaxis of wounds." Undoubtedly real advances will be made in this field as a result of the present world conflict.

Mobile Medical Units

The rapidly moving motorized units, made up of motorcycles, tanks, motor conveyed infantry, fast moving artillery units and dive bombing combat planes, have made the front line a constantly shifting area and have emphasized the need of mobile first aid and evacuation units which can move forward or backward with the rapidly shifting line of battle. The seriously wounded soldiers with cranial, thoracic and abdominal wounds are now best treated by flying them in ambulance airplanes from the battlefield to hospitals far behind the battle lines, where experienced surgical teams can operate under favorable conditions and where postoperative care can be carried out continuously by the same team. Soldiers with severe wounds of the extremities and compound fractures can be conveyed similarly from the mobile first aid stations and evacuation units after control of hemorrhage, treatment for shock, immobilization and chemotherapy, to hospitals for débridement and adequate after care. Lowering of mortality among the wounded undoubtedly will come with the development of rapid transportation from the combat zone to the hospitals in areas removed from combat.

Treatment of Shock

Improved methods of determining early shock, and the prevention of its late manifestations by the use of banked blood, plasma and serum and by restoration of salt and fluid balance, have been of the greatest benefit to the wounded soldier and civilian, both as an immediate emergency measure and in postoperative care.

Débridement

The last two years of the war of 1914-1918, and experience in traumatic surgery in civil life since then, but especially lessons learned in the current conflicts, have emphasized the importance of débridement of the contaminated wound, of packing the wound with sulfanilamide and vaseline gauze and of immobilization of the wounded part by proper splinting or plaster encasement.

Chemotherapy

Although still in the developmental stage, the use of certain drugs of the sulfonamide group, for combating pyogenic infection in wounds and infection of the blood stream, already has been demonstrated to be one of the great therapeutic advances of the decade. Application of

these drugs locally in the wounds, and administration by mouth and parenterally to the wounded soldier and civilian, is now being generally adopted. For the anaerobic, gas producing infections, compounds that liberate oxygen, such as zinc peroxide, as well as the serum from animals immunized to these organisms, are saving lives and limbs of those whose wounds are contaminated with these anaerobes. The general administration of tetanus toxoid to the troops when inducted into military service is proving even more effective than immunization with tetanus antitoxin in preventing tetanus among the wounded.

Treatment of Compound Fractures

During the recent Spanish Civil War, because of the lack of traction apparatus and of ordinary hospital facilities for both civilian and military wounded, the so-called closed method of treating compound fractures was adopted by the Spanish Republican Army. This method, originally advocated and practiced by H. Winnett Orr of Lincoln, Nebraska, and known as the "Orr method," consists of thorough débridement of the soft parts and bone ends, reduction of the fracture, packing the wound open with sterile vaseline gauze or plain gauze and completely immobilizing the reduced fracture by means of a circular plaster-of-paris encasement or cast. The present improved method is (1) meticulous débridement of the soft parts and bone ends; (2) thorough irrigation with sterile salt solution, (3) application of 10 to 12 gm. of sterile sulfanilamide powder in the depths of the wound and between the bone ends, with vaseline gauze packing, (4) accurate reduction of the fractured bone or bones under roentgenoscopic control, and (5) immobilization of the reduced fracture by circular plaster encasement.

PREVENTION OF INFECTION

The Committee on Surgery and the Committee on Chemotherapy and Other Agents, two committees of the Division of Medical Sciences of the National Research Council, have made the following recommendations for the prevention of infections in wounds and burns.* Such minor editorial changes as shifting of center headings to side headings have been made. The beginning and the end of the virtual quotation are each indicated by three asterisks.

* * *

All too frequently under the stress of emergency conditions the ordinary practices which prevail in wards containing surgical patients following which single asterisks appear in these recommendations have been changed from the form which they had in the original directive; these changes have been made to accord with more recent experience. A. O. W.

tients are forgotten or may be neglected and avenues for the propagation of infection are opened. It is because of this that the following suggestions for the prevention of infections in wounds and burns have been drawn up. These suggestions are based upon the latest practical and scientific information that is available. It is realized that these suggestions may be considered Utopian in their scope; nevertheless they represent a goal, the achievement of which should be attempted if the best results are to be obtained

I. General Principles Governing the Prevention of Infection in Wounds and Burns

- A. In wards containing burned patients (all burned patients should be segregated) dressings should be done at least one hour after the bed linens have been changed, and the floor swept. In wards containing wounded patients, a like period of time should be allowed to elapse before wounds are dressed
- B All dirty bed linen should be bagged as it is removed from the bed Under no circumstances should bed linen be thrown upon the floor.
- C During the dressing period, traffic in the ward should be reduced to a minimum All patients should remain in bed and the ward doors closed, if such is possible. The windows should be closed.
- D All dressers and assistants must be adequately masked and gowned, and the hands of the dressers should be scrubbed for ten minutes before beginning the day's dressings. Regular dressing schedules should be made out and posted for each day of the week and in every ward containing casualties
- E. Dressers or their assistants who are suffering from upper respiratory tract infections such as common colds, grippe, sore throats, or from any infection of the hands or fingers should, if possible, be relieved of their duties as dressers until they have recovered.
- F. All patients should be masked during the dressing period.
- G. Soiled dressings should be immediately placed in a covered container after their removal
- H. All containers, arm baths, urinals, bed pans, apparatus, blankets, linen, etc, should be sterilized immediately after they have been in contact with an infected patient.
- I. If a plaster cast becomes contaminated with infectious material it should be changed unless there is a serious surgical

contraindication. The surface of a cast cannot be sterilized by antiseptic washes. Casts should be moistened along the line of division with a solution of vinegar at the time they are cut. This will keep down the dust and prevent the spread of infection.

- J If the bandage of a patient has to be removed in the x-ray or physical therapy departments this procedure should be done by a surgical dresser. It is better to prepare the patient on the ward so that his dressing does not have to be removed in the x-ray or physical therapy department.
- K. All crusts, pus, strips or pieces of tan, extruded foreign bodies, bits of tissue, or any other infected material should be placed immediately in a suitable covered container and disposed of in a proper manner.

Preoperative Use of Crystalline Sulfanilamide in Wounds

Recent experiences have demonstrated beyond doubt the value of the application of crystalline sulfanilamide to wounds which are awaiting débridement. There is no indication that such a practice adversely affected later surgical treatment and there is every indication that it prevents the development of infection in contaminated wounds.

Hence it is recommended that crystalline sulfanilamide be applied liberally to all wounds as soon as practicable after they have been incurred.

Nothing in this directive shall be construed as an excuse for not débriding wounds as promptly and as widely as time and circumstances permit. Oral sulfadiazine therapy 4.0 grams should be given or taken at once following wounding according to instructions on packet.* Subsequent doses of sulfadiazine will be administered during the preoperative period only at the discretion of a medical officer.*

Care of Wounds

1. As soon as possible after injury the wound should be débrided. Major blood vessels and nerves should not be sacrificed. All necrotic skin, fascia, and muscle must be excised. The wound should then be thoroughly irrigated with sterile physiological saline solution.

2. Following this not more than 10 grams of crystalline sulfanilamide shall be placed in any one wound or 20 grams in all wounds of a single patient.

3. Wounds should not be closed primarily except for superficial flesh wounds which, when débrided and sprinkled with sulfanilamide, may be closed at the discretion of the operator if seen and operated on within six hours from the time of injury.
4. Regardless of the number of hours a casualty is seen following wounding, if the wound has been treated with crystalline sulfanilamide, débridement should be carried out.
5. Following débridement, irrigation, and the local use of sulfanilamide the area should be covered with vaseline gauze and carefully dressed.
6. All wounded men should receive a "booster" dose of tetanus toxoid

Intra-abdominal Wounds Leading to Perforation of Hollow Viscera

1. Institute shock therapy. Give (1) morphine; (2) keep patient warm; (3) give plasma.
2. If an expert anesthetist is not available use only open-drop ether anesthesia. Spinal and intravenous anesthesia are borne badly by these patients.
3. Scrub abdominal wall with soap and water protecting the wound or wounds of entrance with moist sterile gauze soaked in crystalline sulfanilamide
4. Excise wound of entrance and pack wound with crystalline sulfanilamide. Enter the abdomen through a separate incision.
5. Remove gross fluid soiling of the peritoneum by suction possible.
6. Suture perforations of the bowel avoiding resections unless absolutely necessary. Close intestinal wounds transversely to protect the size of the intestinal lumen
7. Inspect the entire bowel for more than one point of perforation
8. Control all bleeding points in the mesentery.
9. Introduce 6 to 8 grams of crystalline sulfanilamide into the peritoneum before closing
10. Drainage must be left to the individual officer, but remember:
 - (a) You cannot drain the entire peritoneal cavity.
 - (b) Drainage interferes with the healing of intestinal lines. Avoid it unless absolutely necessary.
 - (c) If soiling of the abdominal wound has occurred after operation, drain the wound. Place sulfanilamide in the abdominal wound before closing the peritoneum.

11. Continue sulfanilamide therapy (1.0 per cent solution in physiological sodium chloride) by a parenteral route—150 cc. every six hours for from four to seven days after operation, depending on the condition of the patient.

Sulfanilamide is the drug of choice. To prepare sulfanilamide for parenteral use, add 10 grams of crystalline sulfanilamide to 1000 cc. of very hot physiological saline solution, boil for five minutes to sterilize, cool to 37° C., and administer this solution by the subcutaneous route.* Make such solutions up freshly each day. It is to be given under the fascia of the lateral side of the thigh.

Dosage = 150 cc. of 1 per cent sulfanilamide solution (1.5 grams) subcutaneously q six hours for four to seven days following operation. If the patient's condition warrants it, the dose and time of administration may be reduced. [Change to an oral sulfonamide when the patient's condition will permit. A. O. W.]

Sodium sulfadiazine may also be used. To prepare, add 5 grams of sodium sulfadiazine to 100 cc. of sterile distilled water (Do not attempt to sterilize.)

Initial dosage = calculated on the basis of 0.1 gram per kilo of body weight. Inject appropriate amount by the intravenous route. *Never give solutions of sodium sulfadiazine by the subcutaneous or intramuscular routes because they are highly alkaline and may cause sloughing of the tissues.*

Subsequent dosage = 0.03 gram per kilo of body weight given as a 5 per cent solution in distilled water at twelve-hour intervals for four days.

Postoperative Chemotherapy of Wounds

Oral Therapy—On the morning after the wound has received definitive surgical treatment, oral sulfonamide therapy should be resumed.

Sulfadiazine is the drug of choice for oral therapy with sulfanilamide the second, and sulfathiazole the third, drug of choice.

Dosage, sulfadiazine, sulfanilamide, or sulfathiazole. 1.0 gram q. four hours day and night for seven days. At this time if the wound is clean and there is an absence of fever attributable to infection, oral therapy should be discontinued. However if the wound is infected it should be continued as indicated.

If the patient is not voiding normally (1000 cc. per day) the blood concentration of the drug should be determined daily and the dose adjusted downwards if necessary. If complete or nearly complete urinary suppression occurs, omit the drug and force fluids orally if possible and intravenously (glucose and water) if necessary.

Local Use.—Crystalline sulfanilamide appears to be the most satisfactory sulfonamide compound for local use at the present time. Sulfathiazole powder, when applied to wounds, tends to cake and may at times act as a foreign body. It is therefore not recommended at the present time.

Open Wounds—The wound area should be sprinkled with enough crystalline sulfanilamide to “frost” it every time the dressing is changed. This local application of sulfanilamide should be continued until the wound is healed or is secondarily sutured and is essential in the preparation of wounds in which skin grafting is to be done.

Penetrating or Through-and-Through Wounds—It is possible to make a suspension of sulfanilamide crystals in sterile physiological saline solution and then to use this suspension as a means of introducing sulfanilamide into deep wounds. The suspension should be made up just at the *time of use* and the syringe containing it should be rotated while it is being introduced into the wound in order to maintain the suspension.

Where gauze drains or wicks are being used, these should be moistened in sterile saline solution and then dipped in crystalline sulfanilamide before being introduced into the wound. The wound then should be frosted with crystalline sulfanilamide before the dressing is applied. Local therapy with crystalline sulfanilamide should be continued at each dressing until the wound is healed.

Compound Fractures Treated by Orr-Trueta Method—Every time the cast is removed, the wound area should be “frosted” with crystalline sulfanilamide after the wound toilet has been made. When the cast is finally removed from the wound the wound area should be “frosted” with crystalline sulfanilamide at each dressing until healing takes place.

Infected Wounds—In wounds already infected or in which infection arises, the purulent or necrotic material should always be removed prior to frosting the wound with crystalline sulfanilamide. This can be accomplished in the case of pus by gentle irrigation with warm physiological saline solution or, if a necrotic crust is present, by the use of irrigation with azochloramid or some comparable solution q six hours until the necrotic material is removed.

The full value of local sulfanilamide therapy is lost if crystalline sulfanilamide is dusted upon pus or necrotic material in the wound because such material contains substances which antagonize the activity of sulfanilamide and its derivatives.

When the wound has been cleaned up, it should be “frosted” with crystalline sulfanilamide at each dressing until it has healed.

If crystalline sulfanilamide appears to be ineffective after a thorough trial, and signs of infection persist, cultures should be made to determine whether an anaerobic streptococcal infection is present. If so, the wound should be treated with "Zinc Peroxide Medicinal," according to the technic of Meleney.

Technic of Wound Dressings

It is not necessary in doing surgical dressings that the hands be encased in sterile rubber gloves. The following method is adequate if carefully carried out.

1. Before beginning the day's dressings in a ward, scrub the hands for ten minutes as you would prior to an operation.
2. Have yourself and your assistants masked.
3. Do the clean dressings first and the most severely infected ones last.
4. In dressing any patient's wounds, clean or infected, be sure you do not further contaminate the wounds.
5. Bandage scissors are usually contaminated. Do not use them except for the removal of bandages.
6. Have a properly equipped dressing cart and competent assistants before beginning your work.
7. Use forceps to remove gauze dressings and to apply them. Never use your fingers in a wound. If forceps, hemostats, and scissors are used (the so-called "knife and fork" technique) the dressings can be done satisfactorily without gloves.
8. Place all soiled and contaminated dressings in a receptacle as soon as they are removed.
9. Irrigate thoroughly with physiological saline solution, all suppurative wounds. Then use crystalline sulfanilamide as discussed in section on the "Chemotherapy of Wounds."
10. Remember that a wound need not be dressed merely because the dressings become moist from serum. Repeatedly opening dressings always creates the opportunity for contamination. Whenever a wound is dressed, finish the wound toilet by sprinkling crystalline sulfanilamide on the wound surfaces.

* * *

SECONDARY DÉBRIDEMENT AND SKIN GRAFTING

Early skin grafting in cases of infected wounds of the extremities, and in cases of burns, has been demonstrated to shorten the time of disability and to prevent the later contracture deformities that are

so permanently distressing and disabling. This is indicated especially if there has been extensive loss of skin.

In the repair of an infected wound, with extensive loss of skin, the closing of the wound takes place by fibrous tissue contracture after a prolonged period of granulation. In order to eliminate the prolonged period of formation of granulation tissue, and the underlying layer of fibrous connective tissue, it is now generally accepted that removal of the indolent granulation tissue and of the underlying fibrous connective tissue layer, down to normal muscle, fascia and, if necessary, periosteum, leaving a circumference of normal skin, is indicated. Following dusting of the new wound surface with sulfonamide powder, a "split graft" or "thick, razor graft" of skin from an opposite limb is applied to the surface of the wound. The graft is held in position by firm, even pressure of cotton wool or sponge and the limb is immobilized.

This same secondary débridement and skin grafting is indicated in cases of third degree burn, in which chronic, granulating surfaces are present. Especially is this true in regions involving joints.

The hemolytic streptococcus is especially unfavorable to the "take" of a skin graft and, for this reason, cultures of the wound surfaces should be made before the secondary débridement and skin grafting operation are done. Preliminary sulfonamide therapy and the dusting of the new wound surface should precede application of the skin graft.

REFERENCE

- 1 National Research Council, Division of Medical Sciences, acting for Committee on Medical Research of the Office of Scientific Research and Development. The Prevention of Infections in Wounds and Burns. Memorandum—Committee on Chemotherapeutic and Other Agents, January 6, 1942, Committee on Surgery, January 17, 1942 (Mimeographed.)

VASCULAR INJURIES

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VASCULAR INJURIES

CHAPTER I

SCHEME FOR TREATMENT OF HEMORRHAGE

REPLACEMENT OF LOST BLOOD

FLUID to maintain blood volume should be supplied immediately. At the time of hemorrhage, the secondary changes characteristic of shock probably will not have taken place. That is, vasoconstriction will have occurred, making the remaining blood available for the heart and vital centers but concentration of blood and leaking of fluid from capillaries to tissue spaces perhaps will not have begun. Therefore, saline solution and especially glucose solution (5 per cent) given instantly will help, for the time being, to maintain blood volume and may properly be used when whole blood, serum, or plasma is not available. Reliance should not, however be placed on them. Whole blood, serum, or plasma should be secured.

Red corpuscles, in the immediate treatment of hemorrhage, are not required. If whole blood is available, it should be given. Otherwise, plasma or serum, whether fresh, stored, or prepared from dry material, may be used. Either serves to maintain blood pressure well above the critical level of perhaps 60 to 80 mm. of mercury systolic. Depending on the amount of blood lost, 1000 cc. or more should be given intravenously over a period of one to three hours.

POSITION AND PROTECTION OF WOUNDED

Loss of heat must be prevented by sufficient dry woolen coverings for body and limbs. However there is no need of more than maintaining the individual's warmth. Excessive heat might well excite vasodilation, making a call on the blood already much diminished in volume.

Elevation of the feet and depression of the head are useful, making the existing volume of blood available, by gravity for the heart and vital medullary centers.

Pain should be assuaged by securing a comfortable position and the immobilization of broken bones. If any drugs are given, let them be opiates.

EMERGENCY CLOSURE OF BLOOD VESSEL

The rough and ready control of a great artery is occasionally possible by direct pressure with the fingers at a point proximal to the wound. If the arterial pulsation can be made out, the vessel must be compressed against a bony part. The femur or pelvic girdle for the femoral artery, the humerus for the brachial or axillary artery, a cervical transverse process for the common carotid artery, are cases in point. It is seldom possible to maintain such pressure for more than an hour or so but, if early aid is available, such pressure is preferable to the use of a tourniquet. Elevation of the wounded part should be secured if possible.

Only in the case of a very narrow wound might the inserted finger accomplish the closure of a vessel, but at the expense of an infected wound.

The tourniquet will be the usual resource for dangerous bleeding (see the following chapter). For obvious bleeding from the vein of a limb, that is, a flow of dark blood, elevation should be used and, when combined with local pressure by a pad of cloth and a bandage, usually will control hemorrhage

CHAPTER II

INJURIES OF LARGE ARTERIES

APPLICATION OF TOURNIQUET

THE materials preferred for this purpose are the Esmarch rubber bandage or rubber tubing, but in default of these, any cloth may be used. A broad, circular area of pressure is desirable (less risk of injury to soft parts) so that several parallel turns of the material may be taken. However it may be difficult to hold the part elevated while such turns are made or removed, so that there is a certain advantage, especially if the limb is likely to be amputated (widespread and destructive wound or crushing injury) in taking a single turn of an unyielding material capable of being tightened by twisting with a stick. If possible, the limb should be elevated to drain it of available blood before compression is made.

The Esmarch Bandage (Fig. 1 A) is a long strip of elastic rubber bandage about 2 inches (about 5 cm.) in width. It is so arranged that when completely wound about a limb a double tape is available at its end for tying it in place. There may be other fastening devices. The bandage should cover an area several inches wide. It is the ideal form of compression when the wound is of such character that there is a prospect of saving the limb. In that case it had better be applied as far away from the wound as possible, or at the point at which the proximal artery can most effectively be compressed. The advantages of such an arrangement are explained below under the heading, "Site of Application." It is not well suited for hurried loosening and reapplication because many turns must be unwound and reapplied on each occasion.

Rubber tubing can be wound several times tightly about an arm or leg in a series of parallel coils. As the last turn is started, the left forefinger of the operator is inserted under it. Then, when the turn is completed, a loop is passed through the space made available by the finger. Thus, the turn is locked and is readily unlocked (Fig. 1 B). The unyielding tourniquet is represented by any strong piece of cloth or even rope. Once passed about the extremity it is loosely tied

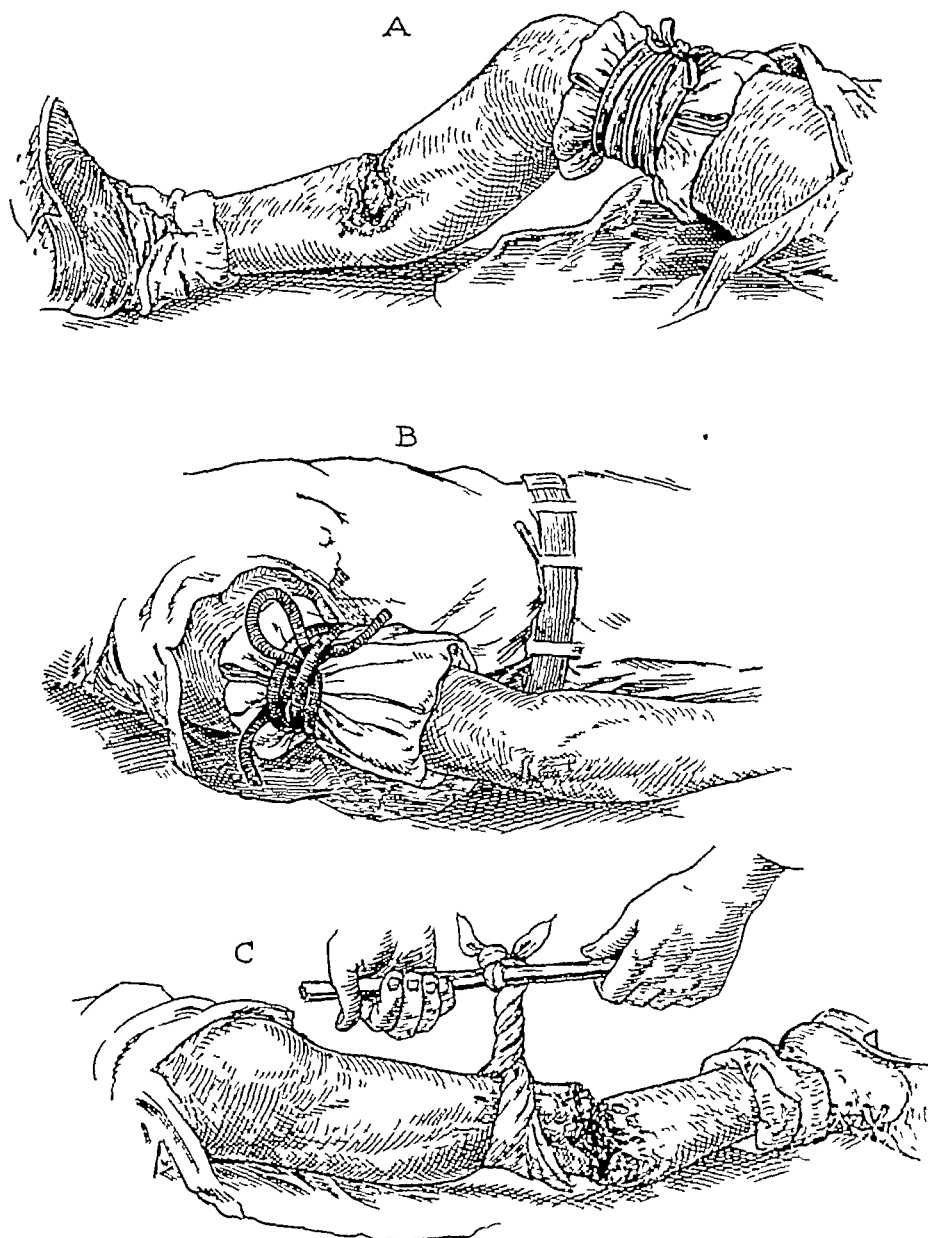


Fig 1—Various tourniquets A, Esmarch bandage is applied for a compound fracture with hemorrhage Ligation of the vessels is planned If ligation is not performed within an hour or two of the application of the tourniquet, pressure should be released at regular intervals B, The use of rubber tubing as a removable tourniquet. This is probably more convenient than the Esmarch bandage as it is easier to apply and remove C, Tourniquet applied immediately above a hopeless injury with total destruction of the blood supply The tourniquet is made of cloth and is applied close to the injury It should not be taken off until preparations for proximal amputation are made

so that a rod or stick can be passed through its knot. Twisting the stick tightens the tourniquet until bleeding ceases (Fig 1, C).

Site of Application

If the compressing device is to be left on for many hours, and especially if the arm or leg seems likely to be lost, the tourniquet had better be applied as close, proximally, to the point of injury as possible. For then, amputation probably will be made proximal to the tourniquet and the injury done by the tourniquet itself will be distal to the operative field. This may be called the application of the permanent tourniquet.

If there is a good prospect of saving the limb, if an early operation seems possible and if the proximal artery is readily available for compression by the fingers, the compressing device should be placed well away from the point of injury toward the root of the limb at the most convenient level. Here, if it is not to be left on for more than one to two hours, it may remain until ligation or repair of the artery. The field of operation then can be prepared and even the exploration and ligation can be made before the tourniquet is loosened.

Whoever applies the tourniquet should plainly label the wounded person with a large Tq

If a period of several, or many hours is likely to elapse before the blood vessel can be controlled, the rule is that the tourniquet should be loosened every hour pressure on the proximal artery being maintained with the fingers. If possible, the tourniquet should be left loose for a minute or two and reapplied as before.

Efficiency of Tourniquet

A tourniquet should be applied tightly enough to control arterial bleeding, but no tighter. If it is too loosely applied (but tightly enough to obstruct venous flow) arterial blood will continue to escape from the injured vessel or vessels and any blood which is able to enter the limb will be retained in the veins or leak into the tissues, a state almost as bad as an external arterial leak. If applied too tightly it will permanently injure nerves and muscles.

VARIETIES OF ARTERIAL INJURY

Complete division of a large artery usually causes immediate death from hemorrhage. There are, however, exceptions. Therefore, if a wound is so situated that arterial injury is likely yet little hemorrhage has occurred, the possibility of serious, delayed bleeding must be kept in mind. Division of a large artery may lead to little, or non-fatal, bleeding under the following conditions

1. In a great, lacerated wound, made by a large, rough missile Here the laceration of both artery and soft parts entangles blood and causes rapid clotting.
2. When an artery is torn by stretching so that the inner coats retract and the outer coats are shredded and aid in securing an obstructing clot.
3. When the escape of blood from the region of hemorrhage is difficult owing to the situation, size, and direction of the wound In that case, a hematoma will form, in which pressure will finally balance arterial pressure and prevent further bleeding (See "Diffuse Hematoma," just below) Local anatomic peculiarities cause arterial division to produce different effects in various regions, such as the neck, axilla, groin or thigh. These are described under "Injuries of Arteries in Special Locations," Chapter III.

Incomplete division is likely to be made by a small missile, so that it often leads to the formation of a hematoma If the companion vein is injured as well, an arteriovenous aneurysm or fistula may result (see page 208) But a lateral wound of an artery tends to gape rather than contract, so that unless the bleeding is checked by the formation of a hematoma, fatal hemorrhage is likely to occur. A bullet wound through a large artery, for instance, may cause a hematoma of large or smaller size to form, depending on the ease with which blood can penetrate fascial planes and intermuscular spaces Yet if the region of the vessel is directly entered by an exploratory incision, the resulting bleeding will be violent.

Type of Injury That Gives Rise to Diffuse Hematoma (Pulsating Hematoma; False Aneurysm)

This is occasioned by penetrating wounds due to bullets or fragments of moderate size The injured artery, as already explained, may be completely or incompletely divided The appearance of the wound gives little clue to the state of the vessel However, a rapidly forming hematoma suggests a widely opened artery and a slowly forming hematoma indicates gradual seepage from a puncture Injury to a deep vessel, such as the femoral artery in Hunter's canal (canalis adductorius), will cause swelling of the whole limb, whereas a wound of a superficial artery will cause a local tumor. If either sort becomes so tense as to compress the root of a limb, the peripheral part, provided it is still receiving blood through the main vessel or from collateral sources, will become swollen and blue, later, cadaveric

All arterial hematomas are likely to exhibit expansile pulsation

but absence of this sign does not bar the diagnosis. Expansile pulsation is not always easy to detect. On auscultation, an intermittent murmur is usually heard, particularly in incomplete sections of arteries which of course are most likely to give rise to hematomas.

(A venous hematoma, even if considerable in size, is less tense than the arterial sort and exhibits no pulsation or murmur. It sometimes forms when a tourniquet has been too loosely applied. Beyond it, the limb is often pale and cold, the arterial pulse feeble, due to pressure.)

The rapid enlargement of any hematoma, hitherto of slow development and moderate size, indicates that hemorrhage from the vessel, restrained for the moment perhaps by an association of clotting and low blood pressure, has suddenly increased. This indicates a serious wound of the artery and is a type of *reactionary hemorrhage*, which is most likely to occur when the individual has regained a normal blood pressure and especially because of some movement or jar. The subject of hematoma will arise again presently when the discussion, more than at present, will be concerned with treatment (page 206)

LIGATION OF LARGE ARTERIES

The operative field should be shaved and scrubbed with soap and water, followed by the application of any antiseptic substance available. As a rule, it will be possible to find the injured vessel within the wound itself. Therefore, the excision or débridement is begun with the tourniquet applied. Once the supposed source of bleeding is controlled, the tourniquet should be removed to test the efficiency of the ligation. Excision of injured tissue and foreign material will then be continued as usual.

If the situation of the wound is such that a tourniquet cannot be kept in place while the wound is explored, or if the application of a tourniquet is impossible, as about the neck or in the region of the shoulder or groin, the artery should be sought at that one of the places, evident in Figs. 2 to 11 inclusive, which is immediately proximal to the point of injury. Here it will be exposed and controlled. Directions for control of the proximal artery in particular locations will be found in Chapter III, "Injuries of Arteries in Special Locations."

The incision must be ample and watch should be kept for the great nerves which are likely to be closely associated with the artery and vein. By approaching the vessel proximal to the region injured, a dry field is assured in which an orderly dissection can be carried out.

In this field, the artery is isolated and occluded by a rubber covered clamp or a piece of small rubber tubing. The original wound can now be explored and the injured artery examined. In treating it, the following rules should be observed:

1. A lacerated or severed artery should be clamped and both proximal and distal stumps isolated. Ligate each stump separately.
2. In an emergency, transfix the artery with any sort of stitch available, using any ligature material available.
3. When proper facilities are at hand, tie the subclavian, iliac, and common femoral arteries with tape, tie the superficial femoral, axillary, and brachial arteries with braided silk, and tie any smaller vessels with silk of chromicized catgut.
4. Doubly ligate all arteries on the proximal side.
5. Do not ligate an artery in continuity if it can conveniently be divided (division prevents peripheral arterial spasm and secures the best collateral circulation).
6. Divide and ligate the companion vein.

REPAIR OF LARGE ARTERIES

This should be attempted only when adequate assistance and materials are available and when asepsis can be secured. Especially fine silk and needles are required. Rubber covered clamps or tubing are used to control blood flow during repair.

If a lateral defect in an artery is present, it may be closed by a series of stitches evenly placed and approximating intima to intima.

If an artery is lacerated, the frayed portion should be cut away with a sharp knife or scissors.

End-to-end union should be made by accurately triangulating the two stumps, using silk stay stitches taken through the intima. Then continuous stitches are taken uniting intima to intima, that is, everting the arterial walls. Into the artery proximal to the repair there should at once be injected about 50 mg of heparin and, several hours after closure of the wound, heparin should be administered intravenously by one of the two following methods.

Preferred Method (Continuous Drip)

Introduce a 17 gauge needle into a vein of the arm or leg. Set up a continuous drip of 5 per cent glucose solution. To 1000 cc. of this solution add 100 mg of heparin. The average rate of the drip should be about 25 drops a minute. Govern the rate of the drip by the clotting time (capillary tube method), which should be kept at about fifteen minutes.

Alternative Method (Means of Giving Drip Not Available)

Inject slowly into a vein 60 mg. of heparin. Repeat every four hours. The clotting time should be observed one hour after an injection and shortly before the next injection is to be given, and the dose governed accordingly.

After Treatment

The region of repair should be immobilized, using a splint or plaster if necessary. The extremity supplied by the divided vessel should not be elevated, but should be slightly depressed. It should be protected against loss of heat by being wrapped in wool or cotton. It should not be directly heated.

To stimulate the collateral circulation

1. The body should be heated under a cradle.
2. Sympathetic procaine block (Figs. 12, 13, 14) should be given and, if necessary repeated.
3. If convenient, and if circumstances seem to require it, sympathectomy (Figs. 15, 16) may be performed.
4. Vasodilating drugs such as papaverine (0.03 gm., or $\frac{1}{2}$ grain) or sodium nitrite (0.04 gm., or $\frac{3}{4}$ grain) may be given.
5. Passive vascular exercise by a suction and pressure boot or by intermittent venous occlusion may be used.

TESTS FOR ADEQUATE CIRCULATION

These are used when the principal artery of a limb has been injured or when division and ligation may be required by such complications as pulsating hematoma (false aneurysm) and arteriovenous fistula. They support and amplify conclusions based on the warmth or coldness of a limb, its color whether cadaveric, cyanotic, or pale, and the presence or absence of peripheral pulsations. But they are intended, first of all, to throw light on the question of an adequate collateral circulation, following division of the principal artery just proximal to the point of injury or disease. Several useful tests are the following

1. Elevate the extremity to an angle of about 30 degrees, compress the artery with the fingers just proximal to the point of injury and apply an Esmarch bandage from the fingers or toes up to the point of injury. After maintaining bandage and pressure for five minutes, release the bandage while maintaining digital pressure on the artery. The limb should flush down to the fingers or toes within, at most, three minutes, preferably within one minute.

- 2 Elevate the extremity to an angle of 30 degrees and compress the artery with the fingers just proximal to the point of injury. At some point distal to the injury encircle the limb with an estimated elastic pressure by light rubber tubing, sufficient to obstruct any venous return, or preferably by the cuff of a blood pressure apparatus, at a pressure of 60 mm. of mercury—the limb to remain horizontal as the artery continues to be controlled by the fingers. The superficial veins should become engorged in thirty to sixty seconds
3. Compress the artery with the fingers just proximal to the site of injury. Place upon the clean, dry skin a drop of 1:1000 histamine solution. Needle the skin through the drop to produce a wheal which can be felt with the fingertips. It should appear in three to five minutes. If histamine is not available, make a wheal by the intradermal injection of 0.1 cc. of physiologic saline solution. This should normally persist for the better part of an hour. An earlier disappearance indicates a deficient circulation.

The above tests are not conclusive, partly because digital occlusion of the artery may not be complete.

The following tests are useful when the artery is surgically exposed, so that it can be occluded completely by digital pressure, or by rubber tubing or tape directly encircling the vessel.

1. After occlusion of the artery, pulsation of the distal vessels of the extremity should be detectable.
2. After occlusion of the artery, a histamine flare should be secured on any distal part of the limb.
3. After occlusion of the artery for five minutes, the fingers or toes, previously pinkish, still should exhibit some degree of pinkness.

If all such tests are not satisfactory, repair the artery if possible rather than ligate it, or expect the loss of the limb.

If several tests agree in showing a satisfactory collateral circulation when the artery is occluded, doubly ligate and divide it.

Precede arterial division by procaine block of the sympathetic structures (Figs. 12, 13, 14) if possible, and repeat the block daily for a time following the division.

DIFFUSE HEMATOMA

This results, as already partly explained (page 202), from the wounding of an artery without injury to its companion vein. The wound in the skin and deep fascia always will be small and its direc-

to surrounding muscle, fascia, and often important nerves. Before it is explored, the state of the collateral circulation must be studied by the methods outlined in the preceding section, for the injured artery must usually be ligated and the companion vein divided as well.

The delayed operation will be carried out in the same way as the immediate one, but with special watchfulness against injury to any great nerves presumed to be adherent to the sac; that is, by proximal control of the artery, opening the sac and searching for the injured vessel, with double ligation and division (rarely repair) to follow.

ARTERIOVENOUS FISTULA OR ANEURYSM (VEIN AS WELL AS ARTERY INJURED)

The cause of this important injury is usually a wound by a small blade or a small missile, such as a fragment of metal or glass. A bullet may leave a connection between artery and vein, by partly dividing both vessels. If the artery and vein adhere, there is a direct connection or fistula. If an intervening clot is formed and organized into a sac, the condition becomes one of arteriovenous aneurysm. In either case, there ultimately develops in the veins of the region a pulsation synchronous with the pulse.

Common sites for the lesion are: the base of the neck, where the connection is between the subclavian vessels or between the common carotid artery and jugular vein; the root of any limb or the flexure of a limb, where artery and vein are in close association, are usually enclosed in a fibrous sheath and are relatively close to the surface.

Early signs are very variable. The missile may have been so small that a wound is hardly recognizable. Then the first evidence of trouble, after an interval of days or even weeks, will be enlargement and pulsation of the neighboring veins. If the vessels pierced by a stab or bullet are close to the surface, some bleeding will inevitably occur and, if a hematoma is formed, some degree of swelling as well. But even then there is seldom any indication of the unnatural connection until the pulsating swelling develops.

The local signs of the established fistula or aneurysm are pulsating enlarged veins, with or without a local swelling, and a bruit on auscultation which may be continuous but which will have a systolic accentuation. The enlarged veins are usually deep ones which are pushed up toward the surface, but superficial veins may be obvious as well. In the periphery of the limb, the pulse sometimes disappears but is in any case weaker than that in the corresponding extremity and the blood pressure is lower. The temperature of the skin is often

raised in the vicinity of the fistula but is usually lowered beyond. There may even be gangrene of the fingers or toes.

In the body at large, certain circulatory changes gradually develop. They show themselves within weeks or months, depending on the size of the fistula and its situation. They are most serious in the presence of large communications near the heart. A femoral fistula would ordinarily cause more disturbance than a popliteal one, and a subclavian fistula, more disturbance than a cubital one. There is a general fall of arterial pressure and a rise of venous pressure which is most marked in the affected limb. The pulse rate is elevated. The heart enlarges, subsequently undergoing hypertrophy. It may and usually does, deteriorate and, after months or years, serious decompensation ensues.

Treatment

The object of early treatment is to assist in the formation of a collateral circulation so that when the fistula is ultimately closed—a procedure almost necessarily requiring proximal and distal division of both artery and vein—the extremity will have an adequate blood supply. In the early stages, little actually can be done and, meanwhile, a month or two must elapse before operation can be undertaken. In this period, proximal division of the vein may be helpful but division of the artery invites disaster, for through such collateral circulation as then occurs, blood finds its way most easily through the fistula and not into the periphery. Sympathetic block should aid the collateral circulation.

Operative Treatment

This is undertaken (Fig. 17) after an interval of six weeks to two months and is technically a difficult procedure, because of the mass of greatly dilated veins and the tendency to dilatation and friability of the artery so that avoidance of serious loss of blood during the operation requires skill and great familiarity with the lesion. Provision for retransfusion of the individual's own blood should be made. The exposure must be ample. Once rubber tubing can be passed around the proximal artery a fairly satisfactory control of blood flow is secured, but even then the possible sources of bleeding are often unexpectedly blind and difficult to control. As a rule, four ligations must be performed namely of the proximal and distal artery and the proximal and distal vein. The artery which tears readily must usually be ligated with tape, doubly or trebly perhaps, to secure a broad, even closure. Ideally the lesion should be totally

excised, and rarely a transvenous suture of the false passage is possible

INJURY TO ARTERY WITHOUT ACTUAL OPENING: ARTERIAL SPASM

The passage of a missile close to an artery, or a blow, as in the case of a fracture, sometimes throws the vessel into a state of spasm. There will be no such external bleeding or hematoma as if the artery had actually been wounded. Nevertheless, the arterial supply to the limb will appear to have been cut off.

The limb served by the artery turns white and cold. Not only is it numb to the touch but the individual is unable to move it. The peripheral pulses are absent. Such a state is much like that of arterial occlusion by an embolus except that the limb is not painful. Should exploration be made, the artery appears remarkably shrunken, down to a small fraction of its natural size. However, no local treatment of the contracted artery seems to influence particularly the relaxation of spasm, and a diagnosis is often possible without actual inspection of the vessel.

Treatment

This consists in using all possible means to secure vasodilation in the limb. The body should be warmed by indirect heat or within a cradle, the extremity itself gently massaged but not heated. Sympathetic procaine block should be used. The needle, or needles, may even be left in place for several hours, if supervision is available, and 10 to 20 cc. of 1 per cent procaine solution injected every twenty minutes or so. Vasodilating drugs may also be given, especially if sympathetic block is for any reason unobtainable.

Spasm relaxes slowly as a rule. The peripheral pulses reappear in the course of a few days as color and warmth return.

If, after twelve to twenty-four hours, the spasm remains tight, resection of the closed vessel offers the best chance of securing an adequate collateral circulation (release of reflex vasospasm).

THE NECK

Though the great vessels of the neck may be pierced by missiles entering at a distance, wounds of the anterolateral region, near the sternocleidomastoid muscle, are the common source of vascular injury.

Wounds of the middle and lower parts of the neck, from clavicle to thyroid cartilage, are most likely to involve the *common carotid artery*, the *internal jugular vein*, the *vertebral* and *inferior thyroid vessels*. Multiple divisions are not rare and the vascular lesion is often complicated by injury to the *vagus* or *recurrent laryngeal nerves*, the *trachea* or *esophagus*.

Hematomas due to such wounds are rather common, because of the obstacle the deep cervical fascia offers to the escape of blood. Such hematomas fill the lower part of the neck, rising to the level of the thyroid cartilage and the angle of the mandible, and extending toward the median line, from which they may displace the larynx and trachea. In the case of wounds, seen early, which are bleeding freely, life may be saved and a hematoma induced to form by rough and ready closure of the external wound.

Ligation of Common Carotid Artery and Exploration of Hematoma

Even if the particular vessel injured is not definitely known, the common carotid artery should be exposed in the lower part of the neck, before search is undertaken for the injured vessel.

A longitudinal incision is made over the median border of the sternocleidomastoid muscle, which is retracted laterally, or a collar incision is used (Fig. 2 and legend) and the sternocleidomastoid divided. In either case, the carotid sheath is exposed behind the sternocleidomastoid, the sheath incised longitudinally and a rubber tube or artery clamp placed about the artery, which lies median to the internal jugular vein. Then the hematoma is approached by whatever method is most convenient. The collar incision should be sufficient or the longitudinal incision may be prolonged. Even with control of the lower part of the common carotid artery, bleeding will occur from the distal stump if that vessel is injured. The *vertebral* and *inferior thyroid arteries* (derived from the *subclavian*) are accessible through this same exposure when the common carotid is not the vessel involved. In the case of a carotid-jugular fistula, the hematoma is usually small. The same approach is made.

Even with proximal control of the wounded artery, turning out the great clot and finding the point of injury is likely to result in

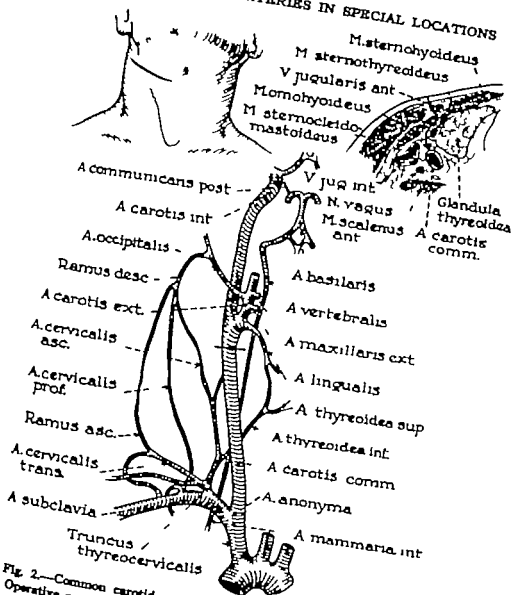


Fig. 2.—Common carotid artery Incision, approach, and collateral circulation. Operative procedure: (1) Place head reclining, neck turned but not over extended toward opposite side. (2) Incise transversely at the level of the cricoid cartilage, centering over the palpable carotid pulse. Divide skin, subcutaneous fat, platysma, and fascia in the same direction. (3) Expose anterior border of sternocleidomastoid muscle. Retract this muscle laterally; the superior belly of the omohyoid forms the inner border of the triangle in which the neurovascular bundle lies. (4) Within the vascular sheath the descending branch of the hypoglossal nerve lies anteriorly the internal jugular vein next, and the common carotid artery slightly behind and medial to the vein. (5) When tying the large vessels do not pick up the vagus nerve which lies closely behind them. The sympathetic chain lies outside of the vascular sheath. The common facial vein, entering the internal jugular at the carotid bifurcation, may have to be tied for better exposure. (6) When the common carotid artery is tied, collateral circulation is established through the vertebral, the opposite internal carotid, and retrograde through the external carotid. Avoid trauma to the bifurcation: use procaine to block the nerve receptors in the carotid sinus.

further loss of blood, and perhaps in a serious fall of blood pressure. This combination exposes the brain to the risk of anemia in case the common carotid artery must be ligated. Normally, however, a retrograde flow, by way of the branches of the external carotid, supplies the internal carotid with sufficient blood to nourish the corresponding half of the brain (the vertebral artery should adequately reinforce the supply through the circle of Willis but does not always do so). Actually, division of the common carotid is less dangerous than combined ligation of the external and internal carotids.

Wounds of the upper part of the neck are most likely to endanger the *external carotid artery*, the *internal carotid artery*, and the *jugular vein*. They involve the parotid region and the mandible. The pharynx is often opened. If the wound is not large and not rapidly fatal, a hematoma is likely to form about the angle of the mandible and submaxillary region. It tends to remain high but spreads inward, displacing the lateral wall of the pharynx. Simultaneous injury of the jugular vein and a great artery may result in an arteriovenous fistula.

Ligation of External or Internal Carotid Artery

Exposure of the hematoma must be preceded by an incision through which the common carotid artery can be isolated and controlled as in injuries lower in the neck. The classical approach to the external and internal carotids is then made by an incision along the anterior border of the sternocleidomastoid muscle from the angle of the jaw downward. The sternocleidomastoid is retracted laterally and the great cornu of the hyoid bone is felt at the bottom of the wound. Just above this point, incision of the deep cervical fascia exposes the external carotid artery and its branches. The internal carotid artery, at its origin, lies posterior to the external carotid. As already explained, ligation of both internal and external carotids should be avoided if possible because of danger to the cerebral circulation; yet even ligation and division of common, external and internal carotids does not necessarily lead to serious unilateral cerebral anemia (effective circle of Willis and supply from the vertebral artery).

THE AXILLA*

If the arm is actually torn off and the axillary artery is ruptured by stretching and laceration, hemorrhage may be slight or absent.

The majority of wounds are perforations due to bullets or irregular missiles. Should the thoracic wall be injured, hemorrhage into the pleural cavity may occur. Usually a large local hematoma, causing a great forward bulge below the clavicle, is formed. Pulsation and

* For the subclavian artery see Fig. 3 and legend

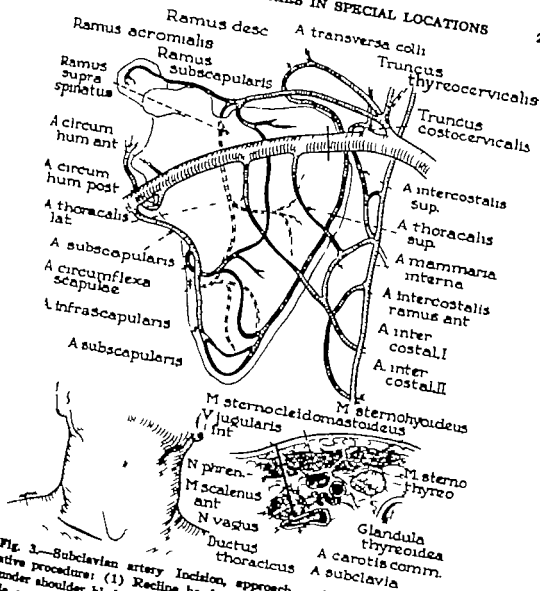


Fig. 3.—Subclavian artery Incision, approach, and collateral circulation

Operative procedure: (1) Recline head and turn to opposite side. Place sand bags under shoulder blades. (2) Incise parallel to, and 1 fingerwidth above, the edge of the trapezius. (3) Section skin, platysma, and branches of the supraclavicular nerves. External jugular vein need not be cut; better ligate between ligatures as nicking it may produce air embolism. (4) Section sternocleidomastoid muscle to its sternal portion; retract internal jugular vein medially. (5) Clear loose fat from the anterior scalenus muscle, define and retract phrenic nerve medially. (6) Sever anterior scalenus muscle with its posterior fascia. Subclavian artery lies immediately behind it, over the apex of the pleura. Subclavian vein lies anterior to scalenus muscle, between it and the clavicula. Brachial plexus lies lateral to the artery partially covered by the anterior scalenus muscle. Abundant collateral circulation through thyrocervical trunk and internal mammary artery; ligate, if possible distal to these.

murmur are usually well marked. The branches of the brachial plexus are very often injured as well, especially the median nerve.

Ligation of Axillary Artery

This is best accomplished by making an incision from the coracoid process, along the lower border of the clavicle, nearly to the sternoclavicular articulation. The clavicular fibers of the pectoralis major

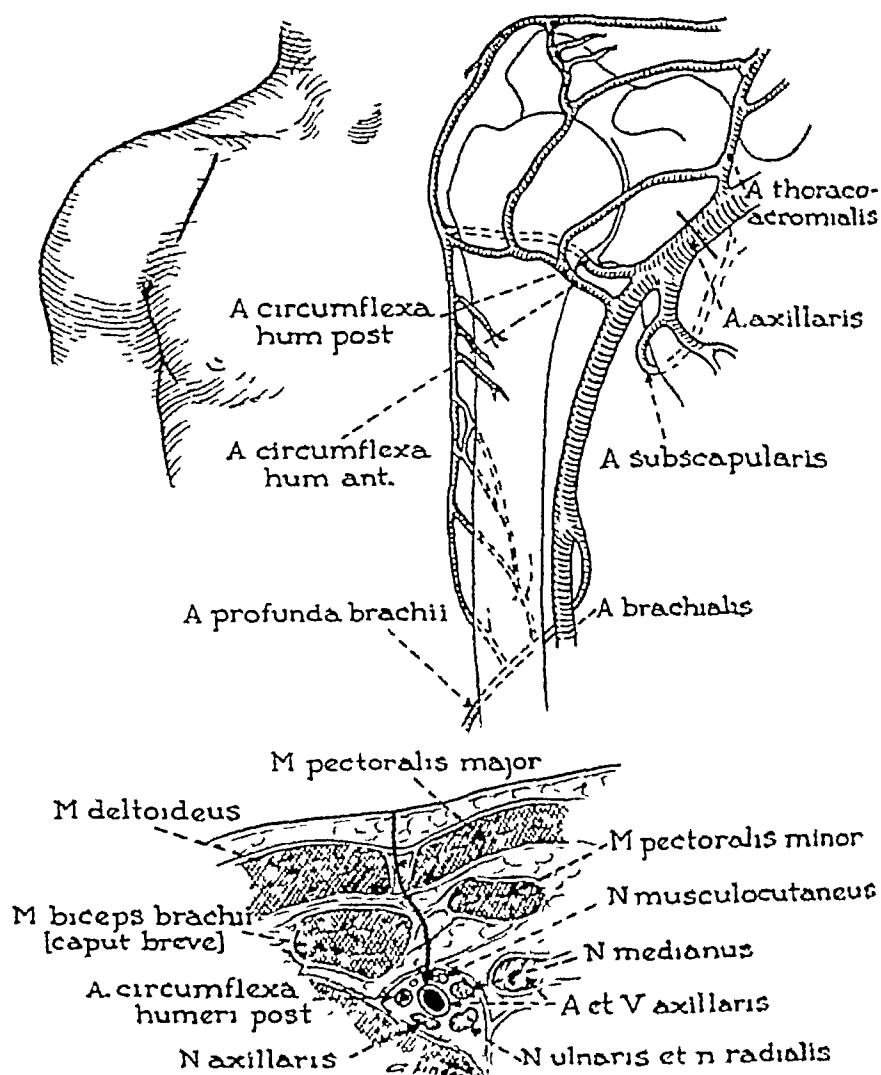


Fig 4—Axillary artery Incision, approach, and collateral circulation Operative procedure: (1) Elevate thorax and moderately abduct arm (2) Incise skin in the groove between deltoid and major pectoral muscles, beginning at clavicle, for a length of 8 to 10 cm. (3 to 4 inches) (3) Retract edges of the two muscles and identify the transverse fibers of the minor pectoral muscle (4) Identify and retract cephalic vein In the loose fat above the free edge of the minor pectoral muscle, the brachial plexus, more medially the axillary artery, and medial to it the axillary vein, are visible Ligation proximal to the origin of the two circumflex humeri and the subscapular vessels is safe, distal to these, gangrene may occur For this reason the infraclavicular ligation is preferred to the customary axillary exposure, during which the dangerous segment of the axillary artery between subscapular and circumflex vessels may be tied

porarily be controlled proximal to the point of injury, after which the hematoma is evacuated and the axillary artery ligated above and below the point of injury. The attempt to repair the axillary artery by suture is seldom made. However, division at its lower end, just below the circumflex branch, carries a considerable risk of gangrene

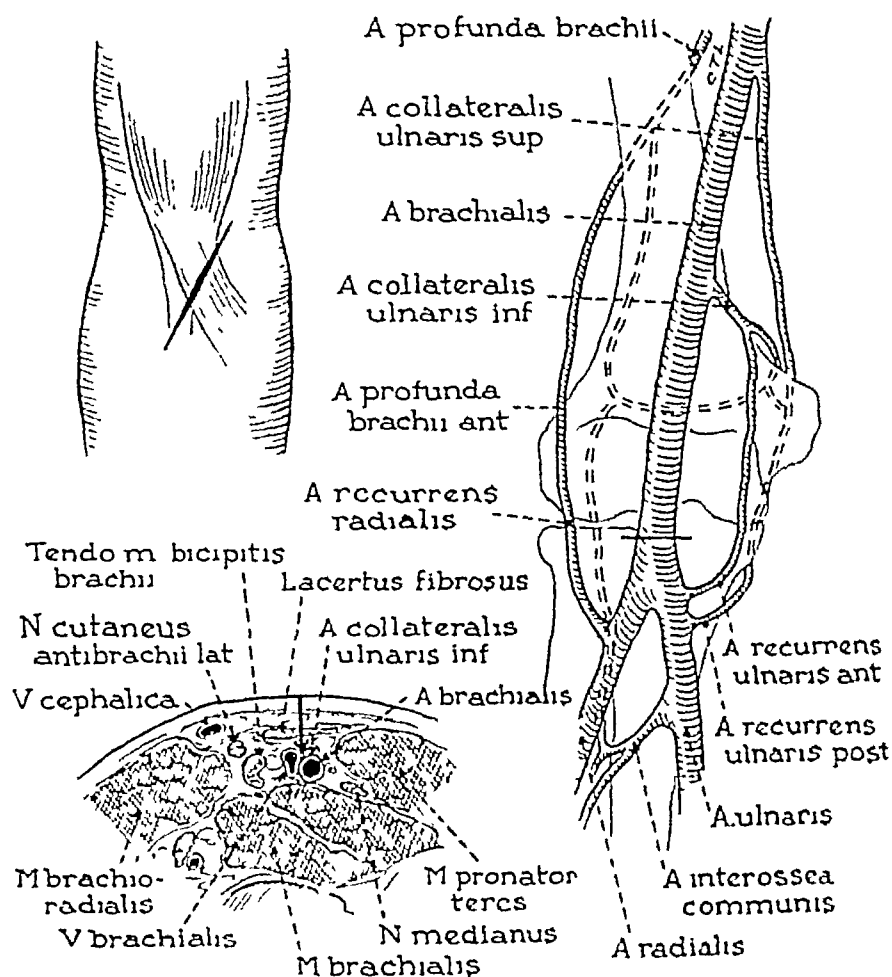


Fig 6—Cubital artery Incision, approach, and collateral circulation. Operative procedure (1) Abduct and supinate arm (2) Bisect lacertus fibrosus through an incision running from the bicipital groove to the edge of the biceps tendon (3) Ligate and cut the median cubital vein (4) The median nerve is medial, lateral to it the vein, between and behind it the artery. If incision is too low the pronator teres muscle is in the way Collateral supply abundant unless the recurrent vessels are destroyed below the bifurcation of the cubital artery

of the hand so that at this point lateral, or end-to-end, suture should if possible be attempted

THE UPPER ARM AND ELBOW

Wounds of the arm, with or without fracture of the humerus, often injure the brachial artery and, in the radial (musculospiral)

groove, the superior profunda artery. Severe external hemorrhage is common and is controllable by pressing the upper part of the brachial artery with the gripping fingers of both hands against the humerus (or by an Esmarch bandage). Wounds by small missiles often lead to hematomas, which diffusely distend the median surface of the upper arm.

Injuries at the elbow, where the artery passes deeply to divide into its radial and ulnar branches, may cause the hematoma to form in the cubital space and extend into the forearm.

Ligation of Brachial Artery

This presents no particular difficulty (Fig. 5 and legend). The proximal part of the artery is controllable by pressure with the fingers while the region of injury is being exposed. The vessel is reached through an incision on a line drawn from the apex of the axilla to the midcubital space. On incising the deep fascia, the biceps muscle is retracted outward. The median nerve lies in front of the artery and vein and must gently be drawn aside by elastic traction.

If the injury is high, the lower part of the axillary artery must be secured to control bleeding. Division of the brachial artery just below the circumflex and above the superior profunda may lead to gangrene of the hand. Otherwise the vessel can be divided with impunity.

In the cubital fossa, the longitudinal incision should cross the space slightly obliquely from within outward. The artery lies close beneath the deep (bicipital) fascia, between the tendon of the biceps muscle externally and the median nerve medially.*

THE GROIN AND THIGH

The femoral artery, especially in Scarpa's triangle (*trigonum femorale*), is frequently wounded. It is again likely to be injured in the popliteal space but in its middle portion, through the greater part of the thigh, it seems to escape injury perhaps because of its mobility.

Great crushing or lacerating wounds often involve the femoral artery in Scarpa's triangle. These wounds are of extreme gravity irrespective of hemorrhage from the artery. The special problem of securing the artery seldom arises in such cases. Most femoral wounds which come to treatment are made by small missiles and result in large hematomas. This hematoma may be principally high and anterior or it may distend the entire thigh beneath the quadriceps muscle, especially if the profunda as well as the superficial femoral artery is injured. The tract of the projectile or a perforating artery may

* For the cubital artery see Fig. 6 and legend.

even lead the hematoma to the back of the thigh. Any branch of the femoral may give rise to a large hematoma, and arteriovenous fistulas occasionally occur.

A high femoral wound cannot be controlled by a tourniquet, but the artery can be compressed by the knuckles or fist against the pelvis, at the level of the inguinal ligament. Early ligation is imperative, because even if hemorrhage does not kill, the hematoma soon becomes so tense as to shut off all circulation to the limb and threaten rapid gangrene.

Ligation of Femoral Artery

Before exploration of a hematoma in Scarpa's triangle (*trigonum femorale*), the *external iliac artery* must be controlled (Fig. 7 and legend). A long incision is made, a fingerbreadth above and parallel to the inguinal ligament, continued toward the flank for a greater or lesser distance, depending on how much of the external and even common iliac vessels require exposure. The aponeurosis of the external oblique muscle is divided in the direction of its fibers. The internal oblique muscle is transversely divided from its inferior border outward and upward perhaps 2 fingerbreadths from its attachment to the iliac crest. The transversalis is similarly divided, care being taken not to open the peritoneum. The peritoneum is then pushed aside mesially, with the fingers, laying bare the psoas muscle and the brim of the pelvis. The external iliac artery lies lateral to the vein and is met with first in the dissection. Soft rubber tubing or an artery clamp is passed about it for temporary hemostasis.

The hematoma is opened for exploration of the femoral artery along a line beginning above, at the midpoint of the inguinal ligament, and the incision is carried downward toward the internal condyle of the femur (Fig. 8 and legend). The great saphenous vein, if encountered, is a good guide to the saphenous opening in the fascia lata. The artery lies lateral to the vein. The fascia lata should be incised downward from the inguinal ligament. The *profunda femoris* comes off the common femoral a variable distance below the ligament (1 to 2 inches, 2.5 to 5 cm) after which the femoral becomes decidedly smaller. These relations are important in exposure of the upper part of the femoral artery, for purposes of hemostasis, when the hematoma is at a lower level.

Ligation and division of the common or superficial femoral artery are seldom followed by gangrene of the foot. The general rule for ligation of large arteries (which see) should be followed.

Because of the possibility of a simultaneous injury of the super-

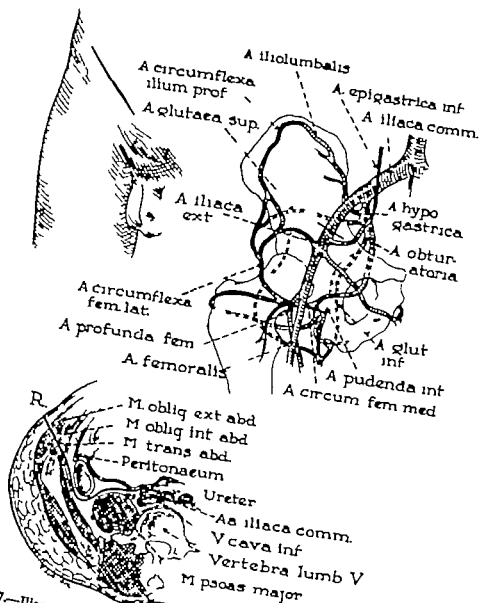


Fig. 7.—Iliac arteries. Incision, approach, and collateral circulation. Operative procedure: (1) Place patient flat on back with sandbag under pelvis. (2) Incise skin from anterior iliac spine to pubic tubercle 3 fingerwidths above and parallel to the inguinal ligament. (3) Tie and cut superficial epigastric vessels, sever in the same direction the sponcturosis of the external oblique, cutting across the internal oblique and transversalis muscles and the transversalis fascia. Do not open the peritoneum. (4) With the two index fingers bluntly dissect off the peritoneum from the psoas muscle and retract it medially and cephalad. The ureter should be retracted with the peritoneum. On the right, the vena cava divides just behind the common iliac artery. The right common iliac vein first lies lateral to the artery then passes behind it to its medial side. The left common iliac vein lies altogether medial to the artery. (5) Collateral circulation following ligation of common iliac artery goes through internal mammary superior and inferior epigastrics, superior hemorrhoidal, the lumbar arteries, and the middle sacral. It is insufficient in sudden occlusions but satisfactory in slowly developing ones or in aneurysms. Collateral circulation after ligation of the external iliac artery is much better. The two important vessels are the circumflex iliac profunda and the deep epigastric, as through them the hypogastric artery can feed the femoral. Tie external iliac above the origin of these two vessels.

ficial and deep femoral vessels, the profunda femoris always should be inspected.

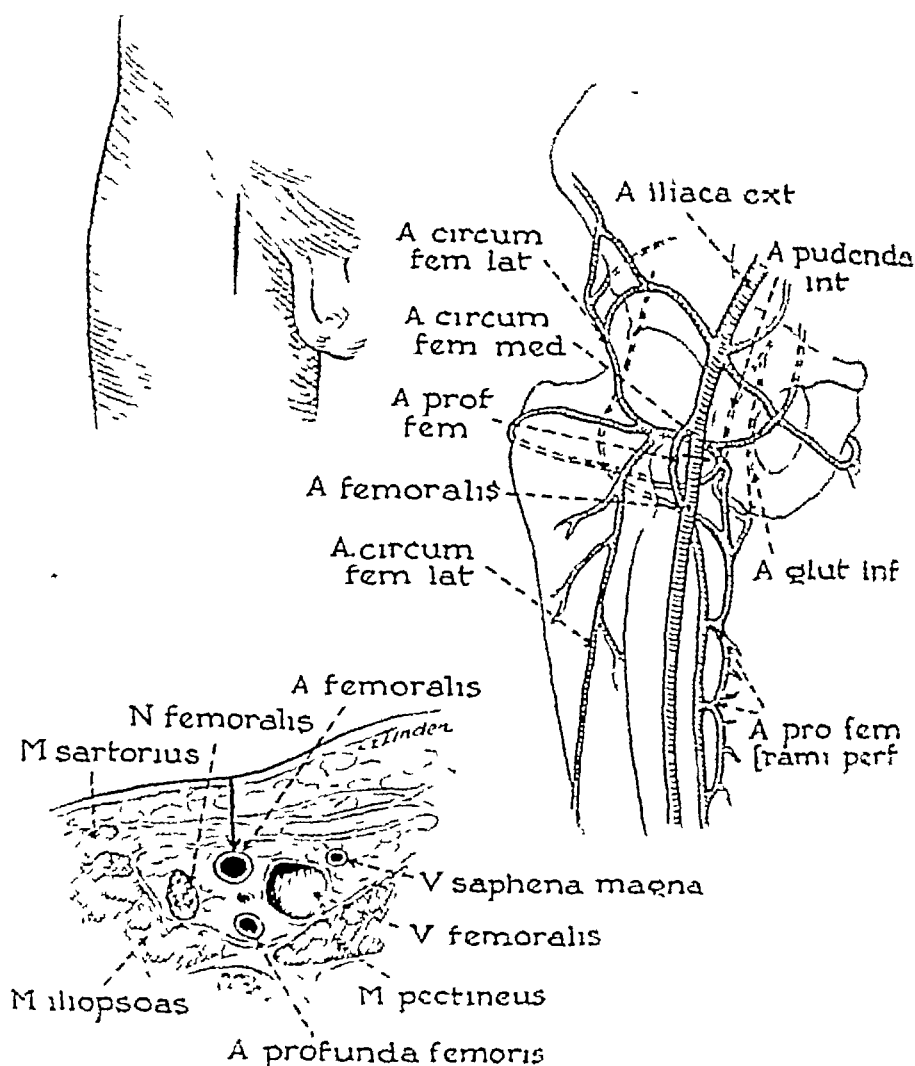


Fig 8—Femoral artery. Incision, approach, and collateral circulation. Operative procedure (1) Place patient flat on back with sandbag or kidney rest to hyperextend the thigh (2) Incise skin from the middle of the inguinal ligament in a longitudinal direction for 10 cm (or alternatively, parallel to and 2 fingerwidths below the inguinal ligament) (3) Identify the inguinal ligament and clear fascia lata of fat and lymph nodes just below it. (4) Incise fascia lata longitudinally. The femoral vein is most medial, next lies the artery, and most lateral the nerve. The major saphenous vein leads one to the femoral. (5) Collateral circulation is established through the inferior pluteal, medial circumflex to popliteal. Ligation below the profunda opens another important channel to the popliteal through a perforating branch. Tie, if possible, below the profunda.

If the vein as well as the artery is injured, each should be ligated and divided at two points, that is, above and below the wound in the vessel.

THE POPLITEAL SPACE

In the case of large wounds, hemorrhage tends to be serious, since the rigid walls of the space keep the wounded vessel open. But most

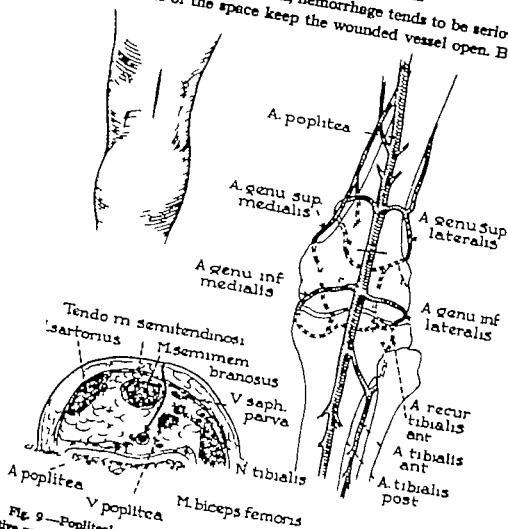


Fig. 9—Popliteal artery Incision, approach, and collateral circulation. Operative procedure (1) Place patient on abdomen with sandbag under knee. (2) Make a generous longitudinal incision through the middle of the popliteal space, incising the fascia. Tie or retract small saphenous vein. (3) In the loose fat, closer to the lateral wall of the space (biceps) the tibial nerve is encountered, isolated with a nerve tape, and laterally retracted; below and medial is the vein, deepest and most medial the artery (4) Collateral supply poor at this level; channels operating on closure of the femoral enter popliteal above the fossa; no large masses of muscles here; therefore, collaterals small. Simultaneous venous ligation able, should be placed above inferior collaterals. Arterial suture preferable to ligation. This and sympathetic block important. Arterial suture preferable to ligation. This caution unnecessary in aneurysms of more than six weeks duration.

patients come to treatment following a small wound and the formation of a hematoma. Such a hematoma rapidly compresses the rather small collateral vessels of the part, causing ischemia of the limb and,

late in progress of the condition, gangrene. Associated injuries of the two branches of the sciatic nerve—notably the common peroneal—are rather common

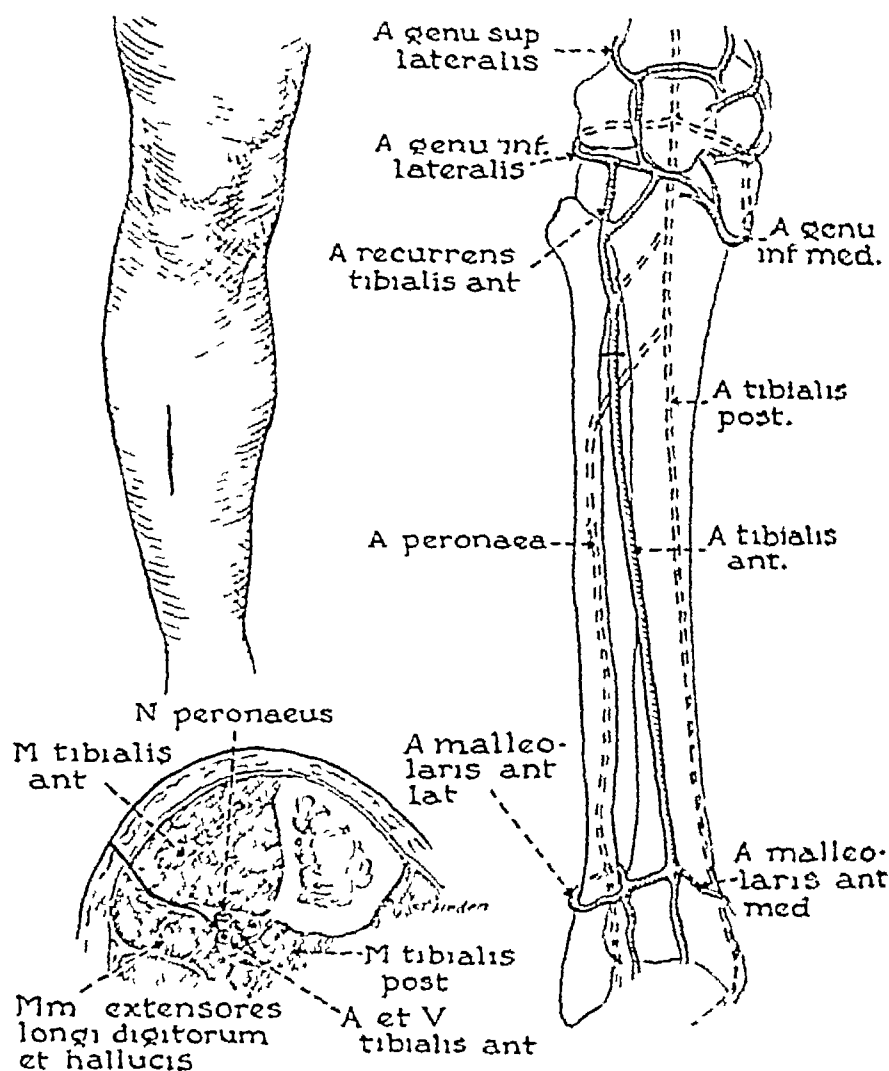


Fig 10—Anterior tibial artery. Incision, approach, and collateral circulation. Operative procedure: (1) Place knee in full extension, patient on back. (2) Incise skin in the middle of lower leg, thumbwidth lateral to lateral margin of tibia. Length 8 to 10 cm (3 to 4 inches). (3) Split muscle, fascia, and carefully identify muscle space between the anterior tibial and the long extensor digitorum muscles. (4) Retract these apart and expose within cylinder of fat the vein, artery, and nerve, which approach the tibia from above downward. There is abundant collateral circulation from the posterior tibial and peroneal arteries. Ligation of both anterior and posterior tibial arteries is safe only if peroneal is intact.

Ligation of Popliteal Artery

This must be preceded by control of the femoral artery. An Esmarch bandage applied to the mid thigh is satisfactory or the artery

may be isolated in the lower portion of Hunter's canal (canalis adductorius)

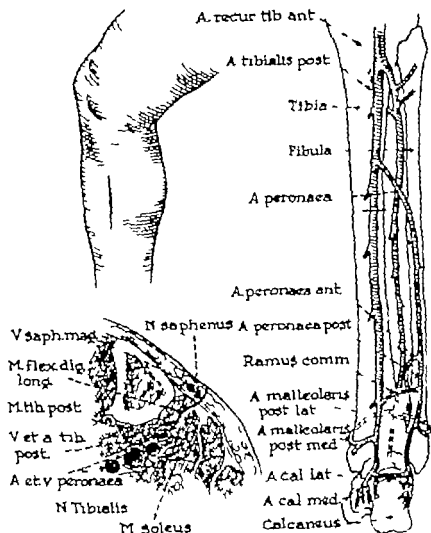


Fig. 11.—Posterior tibial artery Incision, approach, and collateral circulation. Operative procedure: (1) Rotate thigh externally and place sandbag under bent knee. (2) Incise skin for a length of 8 cm. (3 inches) in the middle of the lower leg, 1 cm. medial from the internal margin of the tibia. (3) Isolate and retract the saphenous vein and nerve in the subcutaneous fat. (4) Sever the insertion of the soleus muscle from the tibial shaft and retract the belly of this muscle. (5) Cut the deep fascia longitudinally toward the tibia, the fibers of the flexor digitorum longus and the posterior tibial muscle become visible. The neurovascular bundle lies against the latter muscle. The tibial nerve should be carefully isolated. The peroneal vessels are found deeper in the wound. The main collateral of the posterior tibial artery is outside of the anterior tibial artery the peroneal branch. When both posterior tibial and peroneal arteries are injured, the ischemia of the limb may become quite pronounced.

Incision is made longitudinally along the inner margin of the popliteal space (Fig. 9 and legend) On dividing the deep fascia, the tibial (external popliteal) nerve is seen and retracted outward. The

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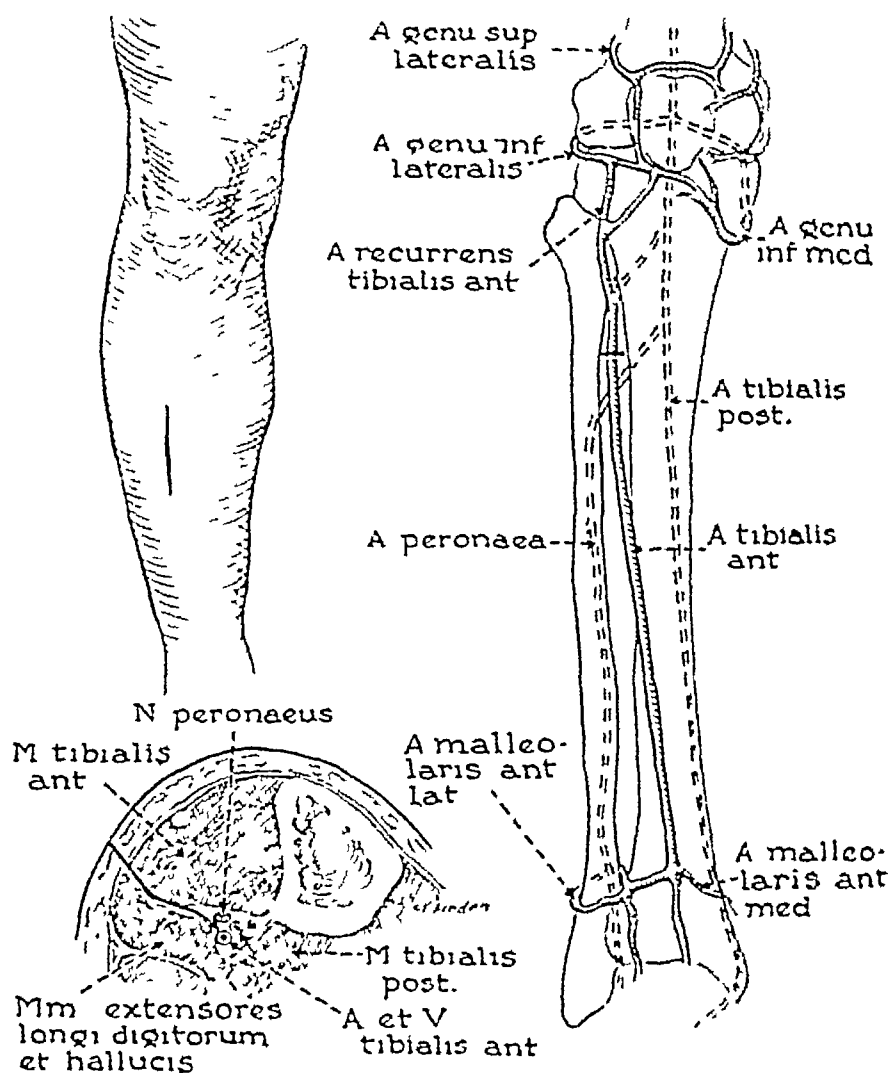


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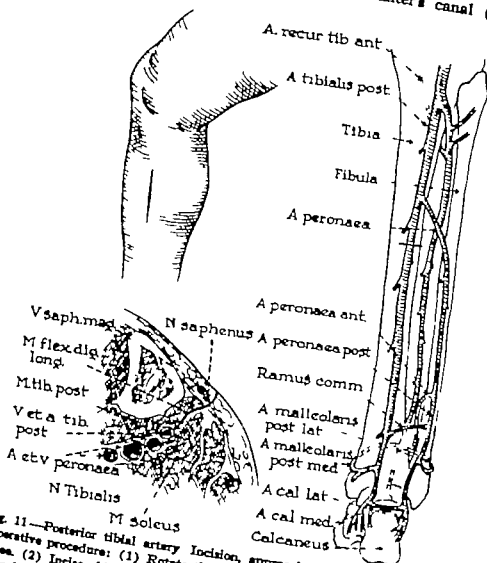


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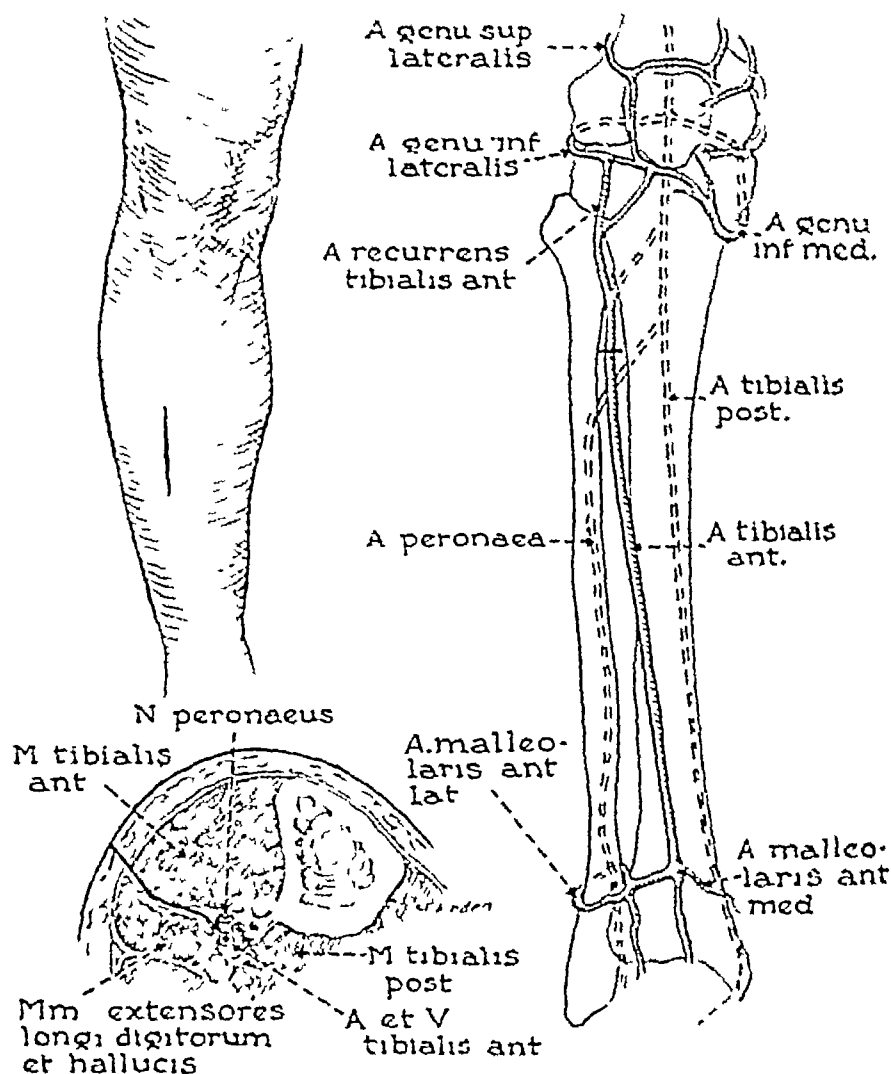


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The most severe types of causalgia are produced by wounds injuring, without destroying, great nerves, especially the sciatic, the brachial plexus, and the median. Sensory filaments, serving the small arteries in particular appear to be irritated, and probably it is these tiny nerves and vessels that are thrown into disorder within the great nerves just mentioned. In these serious cases, pain and some degree of paralysis in the field supplied by the injured nerve are present from the start. Then, gradually, the limb becomes mottled red, shiny slightly edematous, cold or hot, generally paralyzed, atrophied, both as to bones and soft parts and, as a rule, very sensitive to dryness. The altered sensibility defies sensory nerve distribution. The nails are often deformed. Pain usually interferes with sleep.

The minor types arise from small punctured wounds caused by splinters and thorns, from blows, crushes and bites. The region of the lesion is often hypersensitive, the distal extremity or some part of it, dull to pinprick, cool, bluish, and slightly edematous, so that the creases over the knuckles disappear. Pain is always present but varies from case to case. It may be throbbing or darting or aching. It may be both local and distributed over the whole extremity.

The individual is not a malingerer is genuinely upset by his disorder and desires to resume normal activities.

The diagnosis is suggested by the history and physical examination but is confirmed by the remarkable effect of sympathetic procaine block (Figs. 12 13 14). Minor causalgia is relieved, for the moment, completely and, indeed, for several days or weeks, by one injection. Even the patients with severer types of causalgia are greatly benefited. If there is an especially sensitive spot, a scar for instance, local injection of procaine often will give relief in a similar way.

Treatment

The object of treatment is to interrupt the vicious reflex which is believed to be responsible for the disease. The reflex is thought to travel centrally via the sensory nerves serving the blood vessels, enter the cord by sensory roots and connecting with sympathetic outgoing neurons. Thus it can, supposedly be interrupted at its source, or on some part of the great vessels serving the region of injury, or by blocking with procaine or actually dividing the sympathetic supply

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THE LEG

The posterior tibial artery, rather than the anterior tibial, is frequently wounded, especially in connection with fracture of both bones. Often the vessel is temporarily closed by clot in a large wound. In most instances, wounds are made by small missiles and result in hematoma, the diffuse swelling causing great pain. A murmur may be heard. The hematoma is beneath the soleus muscle and lies against the interosseous membrane. Tension rapidly injures the muscle and if infection, especially by anaerobes, is present, gangrene often occurs.

Ligation of Posterior Tibial Artery*

The vessel is approached directly, by a long incision a thumb's breadth posterior to the inner border of the upper portion of the tibia. On dividing the deep fascia, the inner head of the gastrocnemius muscle is drawn backward, exposing the tibia. The soleus is then widely incised about $1\frac{1}{2}$ inches (3.5 cm.) internal to the tibial border. The intramuscular aponeurosis covering the vessels and nerves is split and the tibial nerve and posterior tibial artery, with its venae comites, are revealed. The artery may be ligated and divided without fear of gangrene. Fig. 11 and its legend describe ligation of the posterior tibial artery at a somewhat higher level.

CAUSALGIA; CHRONIC SEGMENTAL ARTERIAL SPASM; REFLEX DYSTROPHY; TROPHIC EDEMA

All such states are undoubtedly interrelated, having a similar fundamental background. All affect an extremity and result from a great variety of wounds and injuries. They are marked by varying degrees of altered sensibility of the skin, by motor weakness or paralysis, by vascular spasm affecting both large and small vessels, by edema, and by atrophy of the bones and soft tissues. Above all, they are marked by pain, persistent and in some cases agonizing. With the severest form of the disease, the pain has a burning character ("caus-

* For the anterior tibial artery see Fig. 10 and legend.

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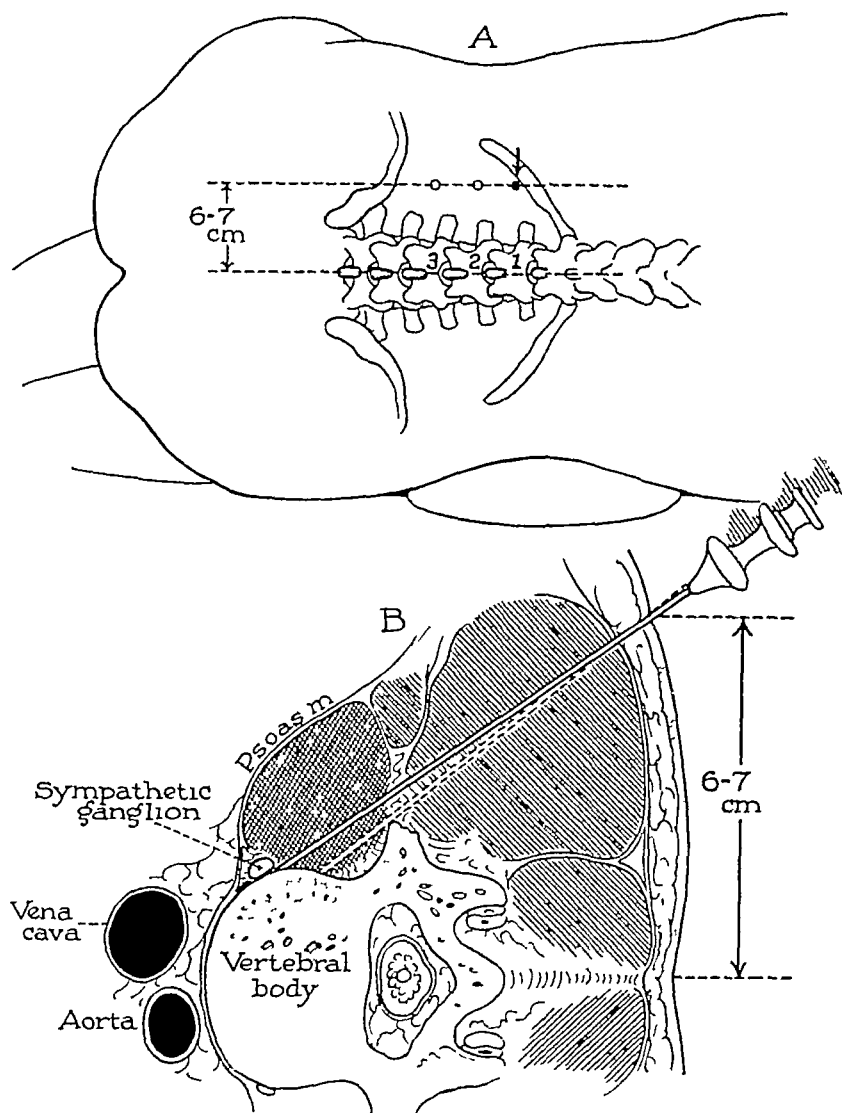


Fig 12—Lumbar sympathetic block. Method I A, The patient is placed on his side, the side of the injection uppermost. A wheal is made 6 to 7 cm from the midline, opposite the interspace between the first and second lumbar spinous processes (preferred region to secure full vasodilatation, the second space may also be used) The wheal falls at the lateral border of the erector spinae muscles and close to the twelfth rib Procaine (1 per cent) is injected into the muscular aponeurosis, which is sensitive B, The 11-cm. (5-inch) needle, with filler, is inserted at an angle of about 30 degrees with the plane of the back. At a depth of about 4 to 5 cm. (2 inches) it may meet a transverse process Passing above or below such a process, it should soon strike the body of a vertebra It is now withdrawn somewhat and redirected, so that again it strikes the body at a more forward point. If the vertebral body has properly been visualized, it should then be possible, by slightly withdrawing the needle and prying it laterally, to cause the point to glide past the body of the vertebra The point should not pass more than 1 to 1.5 cm. (or $\frac{1}{4}$ to $\frac{1}{2}$ inch, which is even less) past the last contact, for at a deeper level it may pierce the aorta (left) or vena cava (right) Always make suction on the needle before injecting Procaine (1 per cent) is injected in any quantity up to 50 cc.

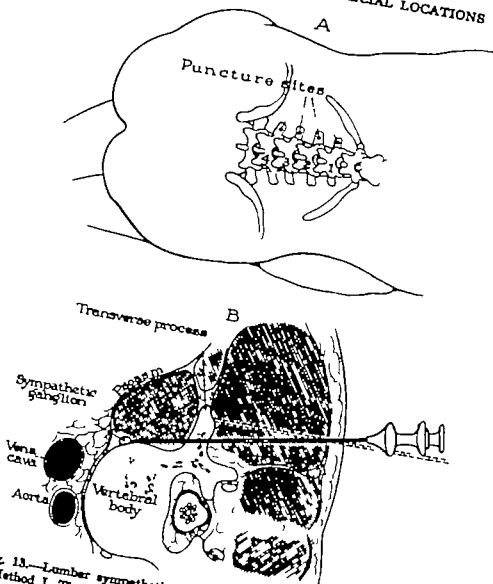


Fig. 13.—Lumber sympathetic block. Method II. A, The patient is placed as in Method I. Three or four wheels are made 2 to $2\frac{1}{2}$ fingerbreadths (perhaps 4 to 5 cm.) lateral to the midline and at the upper border of each spinous process, beginning from above with the first. Each wheel should fall about over a transverse process. The wheels are made with procaine (1 per cent) and the spongy tissue is anesthetized. B, Each needle is first inserted about perpendicular to the plane of the back and should meet a transverse process at a depth of about 4 to 5 cm. (2 inches). It is then withdrawn to the level of the spongy tissue and redirected (higher or lower) more centrally at an angle of less than 25 degrees with the plane of the back. It is passed about 2 to $2\frac{1}{2}$ fingerbreadths (4 to 5 cm.) deeper than the transverse process, so that its point impinges against the vertebra in the retroperitoneal space. When passed in this direction, the point of the needle will remain lateral to the sympathetic chain. Five to 10 cc. of solution are injected through each of the four needles.

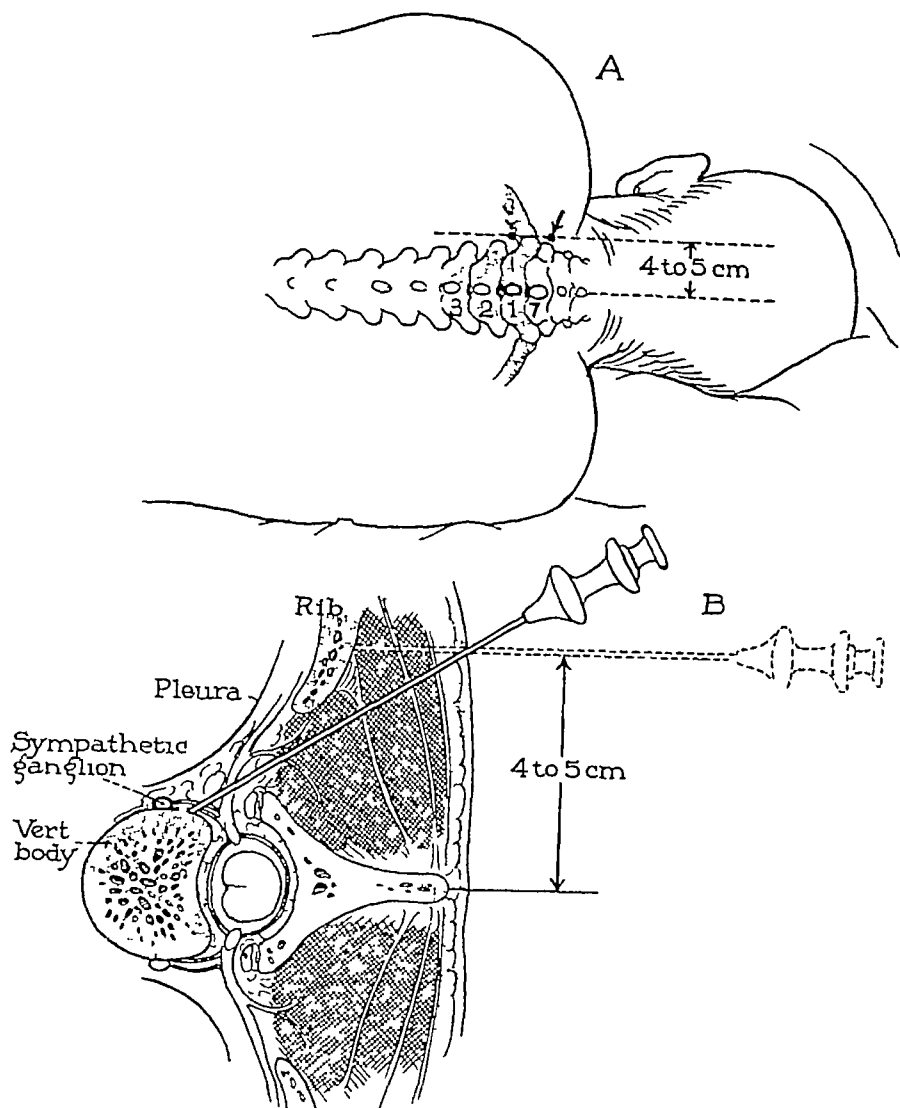


Fig 14—Upper thoracic sympathetic block A, The patient is placed on his side—the head parallel with, or lower than, the neck—the side of the injection uppermost. A spot is selected 4 to 5 cm lateral to the midline, opposite the seventh cervical spinous process (for the injection above the first rib). A wheal is made and the muscular aponeurosis is anesthetized B, The 9-cm (4-inch) needle is introduced at an angle of 25 to 30 degrees with the plane of the back and somewhat caudad. It may strike, at a depth of 3 to 4 cm ($1\frac{1}{2}$ inches), the first rib or the transverse process of the seventh cervical vertebra. In such a case it must be redirected higher (more cephalad) or lower (caudad) as the case may be and at a point perhaps 2 cm ($\frac{3}{4}$ inch) deeper than the rib should strike the side of the vertebral body. The needle must then be directed in a slightly more lateral direction so that it scrapes by the vertebral body. In any case, it must pass within about 2 mm (say $\frac{1}{16}$ inch) of the body lest the point enter the pleural cavity. Its point is passed about 1 cm ($\frac{3}{8}$ inch) past its last contact with the vertebra. Suction on the needle must always be made, to exclude bleeding or entry into pleura or lung. On injecting 10 cc of procaine solution, vasodilatation should be rapid and Horner's syndrome is to be expected. Should procaine be injected into the lung, the patient tastes it (salty flavor) very quickly.

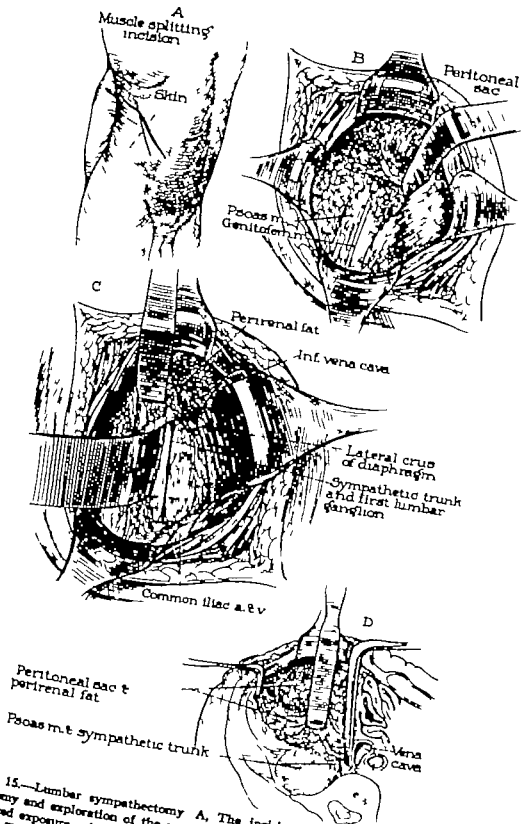


Fig. 15.—Lumbar sympathectomy A, The incision is practically that for a phrectomy and exploration of the ureter. It is principally muscle-splitting, but a good-sized exposure calls for transection of some fibers of the internal oblique muscle. B, The muscles are separated, the peritoneum is pushed forward, and the great posterior muscles are exposed. The genitofemoral nerve is a landmark. C, By depression of the psoas muscle, the sympathetic chain is seen hugging the vertebral bodies. It is pinkish, variable in size. Two ganglia usually are seen, the second and third often being fused. Rami enter the first from above but enter the third from below. D A cross-section to show how the great median vessels are retracted to expose the sympathetic.

to the limb (Figs 12 to 16 inclusive) In the milder conditions, temporary interruption of the reflex may have a long-lasting effect.

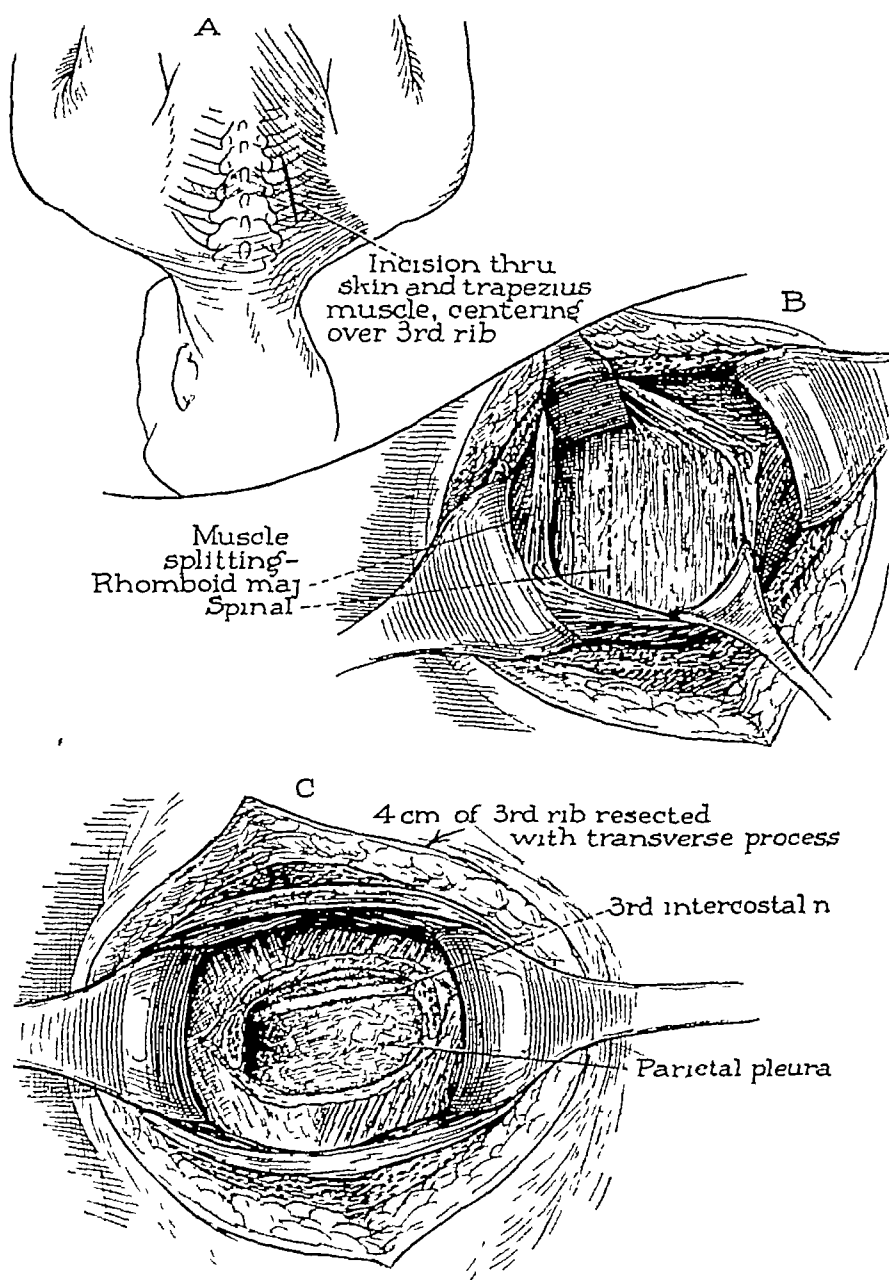


Fig 16, I—Upper thoracic sympathectomy A, The incision, centering on the third rib B, The muscles median to the scapula are partly divided and partly split. C, The third rib resected, exposing the third intercostal nerve The proximal stump is to be rongeured away, and part of the articular process Great care must be taken not to tear the parietal pleura

Rarely one treatment by means of sympathetic procaine block will actually cure, but a succession of blocks, at longer and longer inter-

vah, often does so. Those familiar with the disease use local injection of procaine at the sensitive "trigger point" of the lesion in a

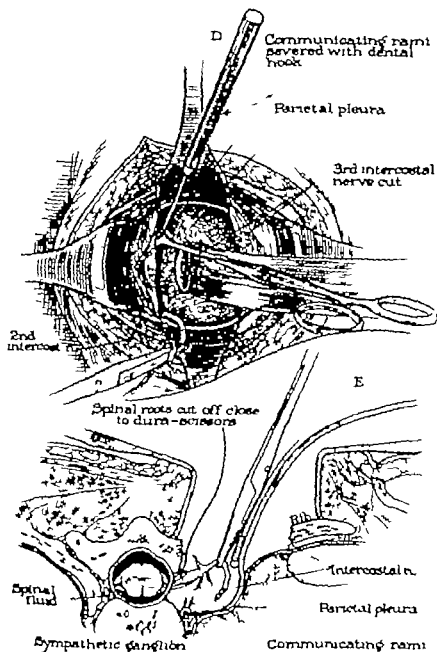


Fig. 16, II.—Upper thoracic sympathectomy D, The second intercostal nerve has been isolated and divided. Traction has been made on the third intercostal nerve and the rami picked out and broken off with a dental hook. E, The motor and sensory roots proximal to the sensory ganglion are divided with scissors. The same is done for the second intercostal nerve.

The last step, division of the sympathetic chain below the third ganglion, is not shown. Care is taken not to disturb the first ganglion and its rami, when the upper part of the chain is turned up.

similar way. Periarterial sympathectomy, which should mean removal of attendant veins and all perivascular nerves, is occasionally and unpredictably successful. It should be directed, if possible, to the artery immediately serving the region of the initial lesion.

For serious causalgia, and as a last resort in minor causalgia, sympathectomy is usually remarkably effective. It had better not be used until any local suppurative lesion is healed (danger of mediastinal or retroperitoneal sepsis, from disturbance of lymph channels carrying infection). Injection of alcohol into a great nerve, in the case of an injury of the median or sciatic, has been tried with success. Ninety-five per cent alcohol is used. Just why this should not set up a new disorder is not clear. On the whole, less traumatizing methods are to be preferred.

ARTERITIS: SCALENUS SYNDROME

Occasionally an artery becomes subject to an inflammatory, irritating lesion of its wall. The cause of such a lesion, other than in relation to the first or a cervical rib, is obscure (distant infection plus injury?) In some instances the wall of the artery is thrown into spasm exactly at the point of irritation and, if the spasm persists long enough, thrombosis may occur. When the subclavian artery (and lower part of the brachial plexus) are compressed in the angle between the scalenus anterior muscle and the first, or a cervical rib, the vessel may be thrown into spasm at that point. If the artery is not actually compressed, it may be affected in another way, that is, it may appear normal or actually dilated as it passes over the rib but be thrown into spasm some distance beyond. This latter type of spasm is probably caused by an irritation, in the subclavian region, of vasomotor nerves which pass beyond and govern some part of the brachial artery.

The effect of such arterial spasms is to diminish or abolish the peripheral pulse. In some instances, the artery undergoes thrombosis and organization; in others it does not. Gradually, rarely suddenly, in the case of a scalenus syndrome, the radial pulse disappears, the hand becomes cool, some of the fingers perhaps become cadaveric in color, and power in the forearm and hand is lost. There is likely to be pain in the region of the neck and shoulder; sometimes in the thoracic wall and arm.

The diagnosis rests on the history and physical signs. But arterial embolism or direct injury to the artery must be excluded. If the neurologic signs—atrophy of the intrinsic muscles of the hand especially—are prominent, the principal irritation is to the brachial plexus rather than to the artery or its vasomotor nerves.

Treatment

Approach is made through a low transverse incision over the sternoclavicular region, as much of the clavicular portion of the

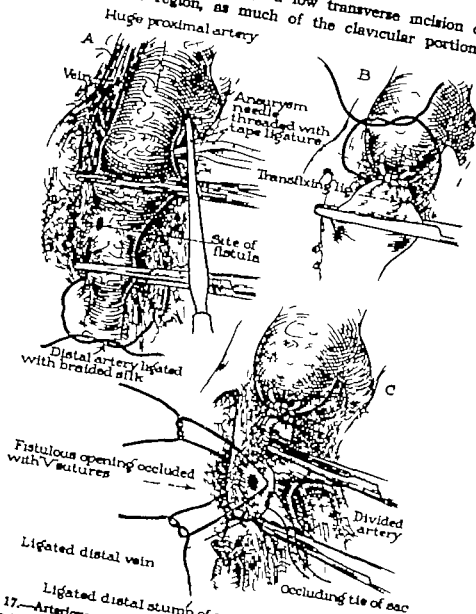


Fig. 17.—Arteriovenous fistula (traumatic) A, The huge easily torn proximal artery is shown with a tape being placed about it. The distal artery is smaller and more easily managed. B, A transfixing ligature is placed around the proximal artery distal to the tied tape. C, The artery is shown ligated and divided proximal and distal to the fistula. The veins are large and have fairly thick walls. A ligature is placed about the vein distal to the sac, and proximal bleeding is controlled by sutures to occlude the fistula. The arterial portion of the sac is sutured to prevent seepage. Any redundant part of the sac may be removed.

sternocleidomastoid muscle is divided as is interposed, the carotid sheath is displaced medially the phrenic nerve is isolated and the

tendon of the scalenus anterior muscle is exposed. The tendon is now divided, adequate protection being given to the subclavian artery and brachial plexus behind it. These structures should then slide forward and downward, so that they are no longer stretched over the rib. However, if a cervical rib is present, some part of it, including its periosteum, usually must be resected. Care must be taken not to open the pleural cavity.

Should the artery, in any part of its course, be found to be permanently occluded, its closed portion is best resected in order that peripheral vasomotor contraction may be relaxed and collateral circulation encouraged.

ARTERIAL EMBOLISM

This rarely will be encountered among young and vigorous individuals, since it usually results from some degree of cardiac decompensation, especially fibrillation, and following myocardial infarct. Emboli also may be detached from the aorta as a consequence of degenerative changes.

The lower extremities are the usual site and here embolism is most serious, for emboli are much more rare in the arteries of the arm and cause a lesser degree of permanent obstruction and ischemia than in the leg. An embolus is very likely to lodge at or near the bifurcation of an artery, notably the iliac or femoral bifurcation, since the caliber of each of the two branches is less than that of the main stem and the embolus actually catches on the partition separating the two branches. Sometimes the embolus, after lodging for a time, slides off into one branch or the other. This may happen at the aortic bifurcation. In the upper arm, the axillary artery, at a point where it can easily be palpated, is the common site; in the leg, the femoral division at the groin.

The onset of an arterial embolism is very painful, fearfully so in some cases, but since pain is apparently due to ischemia and to spasm of the whole arterial tree, the early symptoms are not necessarily acute. There may be a preliminary coldness and numbness of a limb, even with temporary relief before the final painful episode. Both legs may show some pallid cyanosis, associated with numbness and coldness (temporary lodgment at the aortic bifurcation), after which one will clear up or improve and pain will settle on the other. Any serious embolism causes a purplish discoloration of the toes, foot, or even the whole lower part of the leg. The limb becomes powerless. The peripheral pulses disappear.

The situation of the embolus is determined by the point at which pulsation of the great vessel no longer can be felt. This point usually will be farther proximal than one would suppose, judging by the physical signs. For instance, following within an hour or two after serious pain has attacked the right lower extremity the foot and lower half of the leg will be cyanotic, and coldness will have mounted to the knee. At this time, a pulse will be felt in the common femoral artery for perhaps a thumb's breadth below the inguinal ligament but nothing below this point. Almost certainly the embolus must then be lodged at the right femoral bifurcation. If an individual is young, gangrene is unlikely to involve more than several toes, and perhaps the heel if he is arteriosclerotic, the foot and part of the lower leg.

In the case of the arm, coldness and pallor of the hand, and loss of the pulsations in the brachial artery and below are consistent with lodgment of an embolus in the axillary artery. Even then, gangrene of any of the fingers is unlikely to occur.

Treatment

The success of treatment depends on the promptness of its application. If it is delayed for four hours or more, thrombosis is likely to spread from the embolus into the peripheral arterial tree. Moreover the intima at the point of lodgment will soon be so injured that embolus soon follows embolus. However it is said that parinization* so delays secondary thrombosis that operation may succeed even after twenty four hours following lodgment have elapsed. As soon as the diagnosis is made, papaverine hydrochloride should be given, $\frac{1}{2}$ grain (0.03 gm.) and the dose should be repeated in an hour.

Operation (Fig. 18) consists in exposing the plugged vessel, isolating it, and passing short pieces of small, soft rubber tubing about it, proximally and distally so that blood flow from either direction can be controlled. In the case of the upper part of the femoral artery (a longitudinal incision crossing the inguinal ligament is generally used) the various great muscular branches (profunda femoris) must be included. Two per cent sodium citrate solution should be available and the regulation fine artery needles, threaded with the finest braided silk, for repair. As the various tubes are tightened, a longi-

* Of the standard (Toronto) solution, 1000 units (10 mg.) are added to each 100 cc. of physiologic saline solution. This is given continuously and intravenously at the rate of 25 drops a minute and should secure a clotting time of fifteen minutes. Other solutions and techniques are available. During transport (capillary glass tube method) 50 mg. of heparin, given intravenously every three hours, will maintain a satisfactory coagulation time.

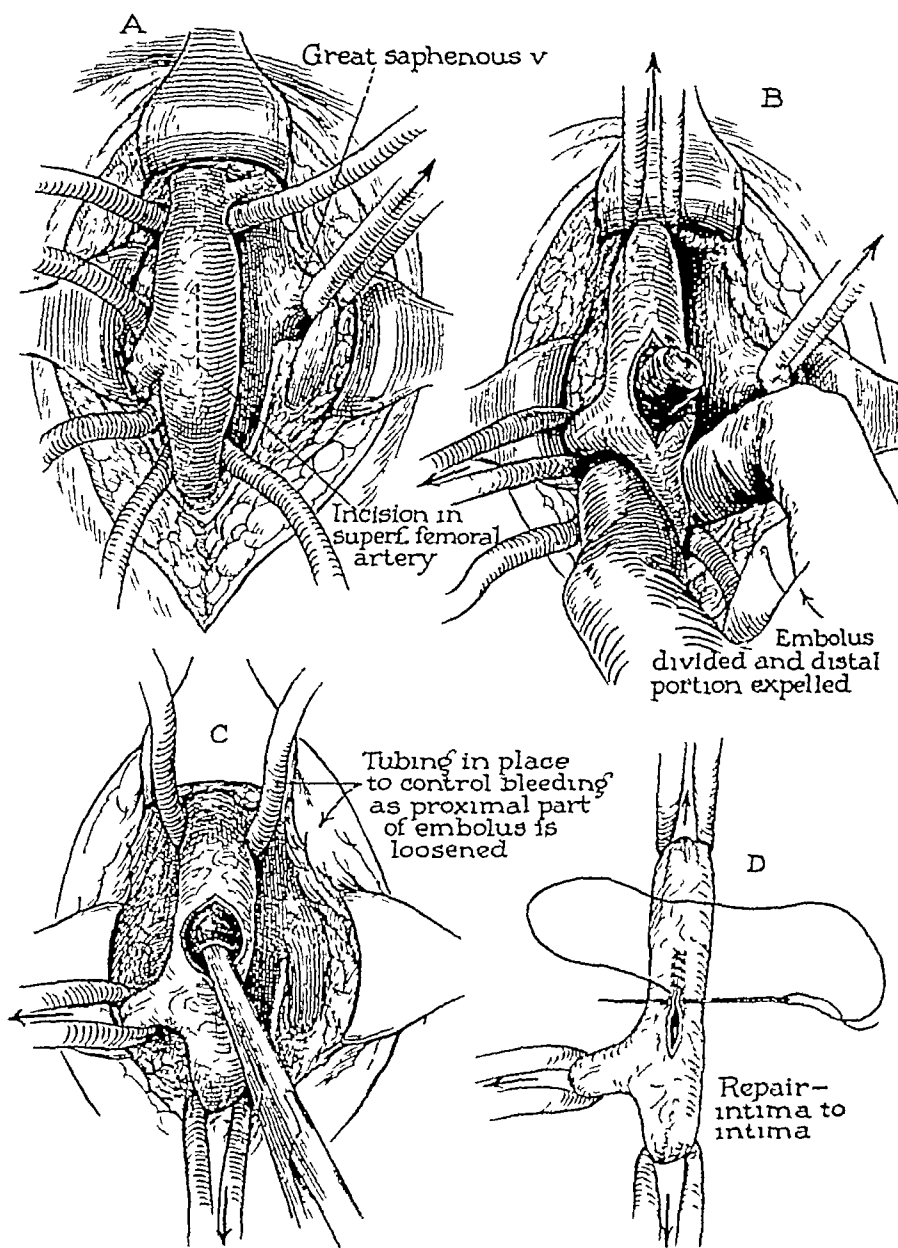


Fig 18—Arterial embolectomy A, The distended right femoral artery containing an embolus is shown. The great saphenous vein leading into the femoral vein (guide to the artery and vein) is shown with tubing about it. Soft tubing controls the femoral artery and its branches. B, While the common femoral artery is controlled proximally, the embolus is squeezed out of the incision. C, Proximal control is released to loosen an embolus still fixed in the artery. D, While tubing is tightened to control bleeding, the artery is repaired.

tudinal incision of the artery is made over the femoral bifurcation. If the thrombus presents, it is sucked out, using a smooth glass tube. If it is lodged above, the upper tube is slackened and some smooth

object such as a uterine sound is poked upward. Milking the clot from above, with the second and third fingers of the right hand inserted retroperitoneally helps to deliver the clot into the incision in the artery. Usually the embolus will come out with a fierce gush of blood, clearing the vessel. Secondary thrombi may be sucked and washed out below. Actually the femoral approach is the one most often used even for emboli at the iliac bifurcation, and at the aortic as well. For with both common femoral arteries exposed and opened, emboli sometimes can be dislodged from below and pushed out by an occasional spurt of blood.

The open artery must be repaired with great care, sewing intima to intima by means of a continuous stitch.

Following embolectomy especially in late explorations (four to twenty-four hours after lodgment) heparinization should prevent thrombosis at the site of operation and beyond.

Nonsurgical Treatment.—When means of operating are not at hand, or when the patient is seen so long after lodgment of the embolus that operation offers no hope of success, every possible means of securing vasodilation should be used. These comprise (1) heating the body under a cradle, (2) administration of vasodilating drugs, (3) sympathetic procaine block (Figs. 12, 13, 14) and (4) use of the suction and pressure boot or intermittent venous occlusion. Meanwhile, the limb must be protected from loss of heat but not directly warmed.

AMPUTATION FOR VASCULAR INJURIES

Since associated injuries such as crushed tissues, extensive destruction of skin and actual or potential infection, are nearly as important as the vascular injury itself rules for amputation on a vascular basis can hardly be laid down. The preferred level for amputation, if the foot must be lost, is 6 inches or 15 cm. (never less than 4 inches or 10 cm.) below the tibial tubercle and if the knee joint must be sacrificed, the most satisfactory amputation is through the condyles of the femur. For the arm, everything is saved that can be saved. Tests must therefore be used to determine at how low a level the injured limb is viable.

Determination of Level of Viability

The problem is always the leg. For in the case of the arm, a system of trial and error is usually possible. That is, the attempt is always made to save fingers, or one finger and a thumb, or the stump of a thumb, or a fragment of hand, and if all the hand must be lost.

any part of the forearm is useful. But with the leg, a crippled foot is useless (save, perhaps, for preservation of the heel in the amputation of Syme). The main question is, then, whether or not an ankle or knee joint must be lost. The following considerations are based on (1) the pulsations, (2) the temperature of the limb, (3) the color, and (4) various special tests.

The Pulsations.—As a rule, a posterior tibial or dorsalis pedis pulsation guarantees that no more than toes must be lost. But absence of both pulses in the foot is not absolutely incompatible with saving it. A popliteal pulse guarantees a stump below the knee joint and, in the absence of such a pulse, amputation below the knee rarely secures a viable, usable stump. A femoral pulse, except for its apparent volume, is of little diagnostic value.

Temperature.—A rough estimate of the temperature of the leg can be made in the following manner. Place the patient in a cool room (about 70° F). Place a heated cradle or warmed blankets over the body. Expose both legs from midthigh downward for twenty minutes. Whatever part of the injured leg becomes cold is not viable. That is, amputation cannot be performed below the lower limit of warmth (see Table 2).

TABLE 2—WARMTH OF PART IN RELATION TO AMPUTATION

Part and Its Characteristics	Indication	
A warm foot (pulsations present)	permits	amputation of toes
A lower leg, cold below the middle third (popliteal pulsation present)	calls for	amputation of the lower leg, 6 inches (15 cm) below the tibial tubercle
A leg cold below the knee (popliteal pulse absent)	calls for	amputation through the femoral condyles
A cold thigh (femoral pulse absent or poor)	calls for	amputation at midthigh, never higher than 3 inches (7.5 cm) below the great trochanter

Color.—Tests by color are unreliable, but some degree of pinkness, as opposed to ivory or cadaveric whiteness, corresponds to the area of warmth. If the limb is elevated, and a white pressure spot is made, the prompt return of color usually indicates capillary circulation. But if the part is depressed, blood merely flows back by gravity into the empty vessels. Much the same is true of the superficial veins. Empty a vein toward the periphery from a valve while the leg is

CHAPTER IV

DISEASES OF ARTERIES

THE occurrence of *thrombo-angitis obliterans*, *arteriosclerotic* deficiency and various states of *vasomotor imbalance* among selected men of military age is unusual, but all may be present among those offered for enlistment, all may be developed in the military service, and all may, if present, interfere with the healing of wounds and other injuries.

THROMBO-ANGITIS OBLITERANS

This is an inflammation of arteries and veins, especially in the lower extremities (rarely in the upper), cause unknown, which obstructs the vessels in a series of attacks. Obstruction is due to thrombosis and organization. Beneath the muscular sponeurosis, that is in the deep vessels, from the femoral down, both arteries and veins are affected. In the superficial parts, the veins alone may be attacked in a form known as "phlebitis migrans," or wandering phlebitis. Sometimes, the disease first involves the femoral artery in which case the peripheral pulses disappear but little pain or tendency to discoloration or gangrene occurs in the toes and feet (collateral circulation by fine vessels) Or arteries nearer the periphery such as the anterior and posterior tibials, and even their finer branches, are separately or simultaneously attacked, in which case the arteriovenous circulation serving several toes and often part of the foot is greatly constricted and delayed, threatening gangrene. Thus the disease may assume a very chronic or rather acute form but usually comes on rather slowly and in a series of episodes.

Clinical Features

The disease attacks men, twenty to fifty years of age. The first symptom is usually intermittent claudication (limp) Walking at a fair pace brings on, always after about the same distance covered, a crampy numb, painful feeling in the lower leg. This is relieved in a

few minutes by rest but returns on renewal of exercise. At any early stage, the toes and foot will usually appear normal or, at the most, the great or other toes will have a slight reddish-blue color and feel cool to the touch. The peripheral pulses will be feeble or absent. One foot will chiefly be affected.

The milder form of the disease may persist in this state for many months or even years, but a wound or an injury, such as dropping some object on the foot, or severe exposure to cold, will precipitate and greatly aggravate the disease.

In a more rapid, serious form, or later, after a mild beginning, coldness, deep redness and edema appear and spontaneous pain may set in. At this time, gangrene of some extent is imminent or actually present. An area beside the great toenail or the outer half of a toe is likely first to exhibit gangrene or ulceration. Both feet are now clearly involved. Elevation causes increased pain and depression seems to relieve but causes edema, a vicious circle, because edema increases the circulatory disorder. Sleep becomes difficult.

Pain and cyanosis seem to be unfavorably influenced by smoking tobacco, especially cigarettes, and by the presence of dermatophytosis. It is rare to see an individual suffering from serious thromboangitis obliterans who is not a heavy or a fairly heavy smoker, and who does not present some signs of infection with the skin fungi. The very bad effect of a wound or injury has already been mentioned.

The course of the disease is seldom toward extensive gangrene, but toward a combination of gangrene and pain which defies relief and demands amputation. Sepsis sometimes originates in local gangrene and seriously involves the leg, but is less common than in arteriosclerotic states.

Treatment

This can best be expressed in a series of general directions for which most of the details need not be presented.

1. Tobacco is to be completely and finally withdrawn.
2. Rest, with good food and surroundings, is to be secured.
3. Special care is to be given the nails and skin, including corns and calluses.
4. Treatment of dermatophytosis is to be given by means of soaks in such fluid fungicides as dilute chlorinated soda, potassium permanganate 1:3000 and by means of ointments.
5. Buerger-Allen exercises are to be performed for periods of half an hour several times a day.

6. Pain is controlled by

- (a) Sedatives other than morphine.
- (b) Discovering the optimal degree of depression of the limb for comfort in sleep.
- (c) Applying dry heat to body and thighs.
- (d) Use of suction and pressure boot or intermittent venous hyperemia.
- (e) Amputation of gangrenous toes.
- (f) Lumbar sympathectomy (Fig. 15)
- (g) Local nerve crushing in the leg.
- (h) Amputation of limb, usually at the knee.

ARTERIOSCLEROTIC DEFICIENCY

This is due to narrowing of the principal arteries of a limb, almost always the leg, by arteriosclerotic thickening plus episodes of thrombosis and organisation. The obstruction is usually incomplete, giving the vessel an irregular caliber. Occasionally an occluding thrombosis occurs, causing more or less marked aggravation of the disease.

Clinical Features

The disease seldom causes symptoms if patients are less than fifty-five to sixty years of age. Men and women are about equally affected. The feet become cold and bear cold badly. Easy fatigue is noticed. The legs appear atrophied and tend to become pale. The soles of the feet may feel "cottony." Intermittent claudication may come on rather suddenly as a result of an accession of thrombosis and obstruction. In that case, some degree of cyanosis usually appears in the toes and forefoot (less marked than with thrombo-angitis obliterans) and spontaneous pain of some degree may set in, especially at night when the legs are no longer depressed.

At this time local gangrene, over a bunion or beside a great toenail or involving several toes, may occur. The gangrene is usually dry and mummifying, but may be complicated by infection. If infection is present, lymphangitis and cellulitis become dangers, joints in the toes break down and local necrosis of toes occurs. Such infections are more frequent and serious in diabetic patients than others.

Absence of peripheral pulses is the rule, but the presence of a pulsation in the foot is nevertheless compatible with arteriosclerotic gangrene. A popliteal pulsation is no guarantee of good circulation in the lower leg, nor is its absence a sign that the leg will be lost.

The foot and lower leg of the affected limb are decidedly cool to

the touch. On elevation for two minutes (to 30 degrees) the toes become cadaveric in color—any remaining pinkness being a good sign—and on depression, the color of the toes slowly turns in half a minute or longer, toward the red or blue.

Treatment

Less improvement is to be expected in arteriosclerotic deficiency than in the presenile form. Especial attention must be given to the care of nails and skin. The nails must be softened by soaking before being cut and should be cut square, not short (to avoid ingrowing nails). Calluses must be sandpapered and corns treated by an expert. The skin should be dried and greased with lanolin after washing. Woolen socks and loose shoes are advised for cold weather.

Buerger-Allen exercises are helpful and tobacco should be withdrawn.

Any area of ulceration or gangrene calls for rest in bed and extra aseptic precautions. Seventy per cent alcohol as a wash is antiseptic and aids in drying up the part. Soaks in the chlorine derivatives and watery iodine solution should be followed by drying with aseptic gauze and the application of a sterile dressing. The leg should not be heated but loss of heat should be prevented.

Local amputations are dangerous. It is better to let gangrene take its course so that the necrotic part is cast off, a process requiring months. And if a toe is actually amputated, the incision should be made within the zone of reaction, not proximal to it.

In diabetic patients, less actual gangrene and more infection is the rule. Treatment of the diabetes should go hand in hand with local drainage, which may comprise amputation of a badly infected toe and even the head of a metatarsal bone. Spreading lymphangitis and a positive blood culture call for emergency guillotine amputation, usually in the midcalf, occasionally in the thigh.

For arteriosclerotic gangrene involving more than toes, amputation below the knee is totally useless, since a useful stump cannot be secured even if healing is effected. When the individual has prospects of leading an active, not an invalid life, amputation through the femoral condyles should be used. For less active individuals the mid-thigh amputation is preferable.

INTERMITTENT ARTERIAL SPASM: RAYNAUD'S DISEASE

This ill-defined state or symptom complex is rare in men, being seen perhaps one-third as often as in women. It is marked by repeated attacks of vasospasm in the digital arteries, the fingers as a

pecially when aggravated by exposure to cold. In fact, chronic peripheral vasospasm may be the background for a good deal of trouble and occasionally, in the legs, for actual ulceration. Hands and feet such as have been described bear cold, damp surroundings badly. Some of the fingers may actually undergo acute spasm, resembling that of Raynaud's disease, on top of the chronic vasoconstriction. In the case of the legs, "trench foot" (see below) is brought on more often than in normal persons; and in the case of both limbs, chilblain.

The chronic vasospasm presumably occurs in the arterioles, possibly the venules, but certainly not in the larger vessels. It is not associated with edema. With it is often associated livedo reticularis (*cutis marmorata*). This peculiar mottling of the lower legs, especially, is due to stasis in the subpapillary plexus. Ulcerations occasionally complicate such conditions.

The diagnosis is made clear by sympathetic procaine block (Figs 12, 13, 14) of any extremity. The foot or hand, as the case may be, is rendered hot and dry. The color is changed from blue to pink, but if the color is patchy, the patches, though altered in tint, often remain.

Treatment

Application of heat to the affected extremities is the wrong way to correct chronic peripheral vasospasm. Vasodilatation can be secured by heating the body or one or several limbs, but most effectively, of course, by sympathetic procaine block. Such a block gives relief in the presence of the constitutional form only momentarily and, if the hands or feet are to be made warm and dry, sympathectomy for each limb must be performed, a considerable series of operations (Figs. 15, 16).

VASOMOTOR REACTION TO COLD: FROST-BITE; CHILBLAIN; TRENCH FOOT

Frost-Bite

This is the most serious reaction to cold and in many ways resembles a burn. Its exact form depends on the dryness or dampness of the atmosphere, the rapidity or slowness of the individual's circulation and tissue metabolism.

Exposure to severe dry cold is likely to cause intense blanching—vasoconstriction—followed by recovery (reactive hyperemia) or gangrene; that is, actual death of the tissue from ischemia.

Exposure to cold, damp air, even at a temperature above freezing, usually causes long-continued vasoconstriction, marked by cyanosis and some degree of edema. Thus it ends in such states as chilblain and trench foot, states of which the peculiarities depend on the exact

exposed, prominent part. It is seen most often among individuals with blue, cold hands and feet and perhaps low metabolic rate, particularly those unable or unwilling to exercise, yet exposed for long periods to damp cold.

Treatment includes cleansing with soap and water and application of mild antiseptic substances. Vasodilation should be secured by heating the body, by gently warming the part, preventing loss of heat by woolen coverings and, if necessary, by sympathetic procaine block. In the event of recurrent chilblain in susceptible individuals, sympathectomy may be required.

Trench Foot

This state of painful pallor, cyanosis, macerated skin and edema occurs when legs have long been exposed to wetness and cold in tight coverings. It occurred often in the course of trench life during the war of 1914–1918. Vitamin deficiency may play a part.

It is due, apparently, to a persistent, obstinate spasm of the smaller blood vessels. Restriction and slowing of the circulation are so serious in some cases that blistering, and even local superficial ulceration and gangrene, may occur. The foot is chiefly involved, but maceration, edema, and cyanosis may include most of the leg up to the knee.

Treatment consists in heating the body but never the legs. The skin should be cleansed with soap and water. Loose, aseptic coverings should be applied. Sympathetic procaine block will do more than anything else to break up the vicious vasoconstriction, but sympathectomy may perhaps be required. The vasodilating mechanisms, such as the suction and pressure boot and intermittent venous occlusion, are likely to be useful. Vitamin B complex may be given freely.

demonstrated that blood is able to flow down a vein, which normally the resistance of the valves prevents

Trendelenburg Test.—The veins are first inspected so that their distribution and degree of distention shall be known. They are then emptied by elevation, following which the leg is lowered. Blood can be seen and felt immediately to distend the veins of the calf. This demonstrates varicosity but fails to tell if any blood flows from the deep veins into the superficial veins, which normally it cannot do. The following test answers that question.

Trendelenburg Test with Constriction about Thigh.—The leg is elevated as usual. Then a piece of rubber tubing or bandage is tightened about the thigh and the leg is lowered. Blood cannot now flow down the superficial veins of the thigh. Therefore, filling of the superficial vessels of the lower leg in five, ten or twenty seconds, must come, through leaking communicating veins, from the deep system. The commonest leaking vessel is the lesser saphenous vein, which empties into the popliteal veins. Any other leaking communicating veins are usually in the upper part of the calf. By applying the constriction at various levels down to a point below the knee, the situation of the principal leaking communicating vein often can be determined.

Should there be an old history of thrombophlebitis, communicating veins are likely to leak and high division of varicose veins will not benefit varicosity. But the state of the deep veins themselves requires no particular study beyond the observation of the color of the foot. For if, in the presence of varicose veins (which add to the load of the deep vessels), the color of the foot on standing is not cyanotic, the deep veins must be functioning efficiently.

Treatment

The treatment of varicose veins by injection alone is palliative. A vein can be sclerosed, for a considerable distance perhaps, and may even remain obstructed, but if the varicose saphenous vein remains open in the thigh, recurrence is inevitable. Injection of sclerosing substances is therefore chiefly useful to do away with local unsightly varicose veins and to follow up high division. Satisfactory sclerosing fluids for local injections are:

- 1 Quinine hydrochloride (0.26 gm.) and urethane (0.13 gm.) in a 2 cc. ampule
2. Sodium morrhuate (5 cc. dose of a 5 per cent solution).

Although opinions differ, a sclerosing fluid is probably most effective when injected into a vein nearly empty of blood.

High Division of Varicose Veins.—This operation (Fig. 19) which can safely be performed under local anesthesia, requires care-

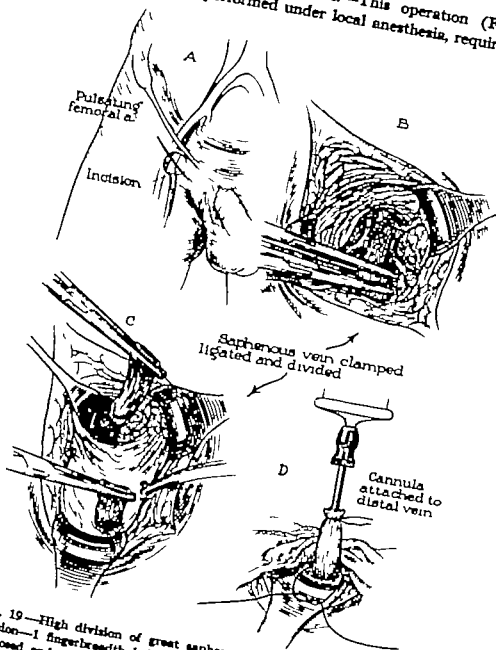


Fig. 19—High division of great saphenous vein. A, Normal anatomy and the incision—1 fingerbreadth below the inguinal ligament. B, Great saphenous vein exposed and clamped, 2 to 3 cm. below the saphenous opening. C, Vein divided and all branches entering the stump divided and ligated. The stump is to be ligated at the level of the fascia lata and a long cuff is left. D Distal stump of the vein ready for injection.

ful dissection at the groin, as shown in the accompanying figure. The great saphenous vein should be divided at its junction with the femoral, at the saphenous opening, and all branches entering the

stump should be divided. This step is taken to make more difficult the reestablishment of a new varicose system through collateral branches. There is usually added to a high division, the retrograde injection of a sclerosing, hypertonic sucrose-saline combination in an amount of about 10 cc. Low division at the knee probably makes the injection from above more locally effective and the sclerosis permanent.

The effect of a high division carries a reasonable hope of permanent cure, so that those who have been subjected to it are fit for active service.

Varicose veins, associated with incompetent communicating veins, are usually a result of former thrombophlebitis, which in any case is likely to cause unfitness for active service (swelling, cyanosis, pain in the leg). The diagnosis is made by performing the second part of the Trendelenburg test. The veins of the calf fill rapidly on lowering the leg while constriction is applied to the thigh. The skin of the calf is often pigmented and discolored. Induration and ulceration are decidedly common.

If surgical operation is used for varicosity associated with a leaking communicating vein, not only should the saphenous vein be divided at the groin, but the leaking perforating vein should be sought. If this is the lesser saphenous, it is best exposed by an incision crossing the popliteal space where it is found beneath the deep fascia, embedded in the thick popliteal fat. If there are good-sized varicose veins and a leaking perforating vein in the calf, a dissection must be made here. This requires skill and perfection of technic because the subcutaneous tissues are often indurated and heal badly. The veins are most safely explored by making long, narrow flaps and thus exposing the aponeurosis. Such operations should be reserved for special occasions and had best not be undertaken in the military service. The individual is seldom made fit for active duty.

VARICOSE ULCER

Ulcers tend at an early stage to be small and directly related to the varicose veins. They are most often situated on the anteromedian face of the leg, one-third of the way from the internal malleolus to the knee. Since early ulcers are usually cured by high division plus injection of varicose veins, such operations are *entirely appropriate* in the military service and carry a good percentage of cures.

Old, deep, or especially extensive and indurated ulcerations are unlikely to be cured by simple procedures and may require, if the

status of the patient is appropriate, excision and skin grafting. Such procedures are not described here.

THROMBOPHLEBITIS IN VARICOSE VEINS

This is very common. It usually begins in a sacculum below the knee and tends to spread upward. The amount of inflammatory reaction about the thrombosed vein is variable. Often the skin is red and the area painful and tender. But breaking down of the thrombosis, much less actual suppuration, is very rare. Although the process may extend to the saphenous opening, it seldom enters the femoral vein or causes embolism. However thrombosis of the varicose saphenous vein is a rather common complication of the condition of femoral thrombosis.

Treatment

An effective ambulatory treatment is the application of an elastic, adhesive bandage from the toes to the upper level of the region of thrombosis. This treatment is most appropriate for a process which has not mounted above the knee. Rest in bed probably cures the disease most rapidly, provided the leg is elevated above the body. An effective method is to elevate the foot of the bed from 4 to 6 inches (10 to 15 cm.) and not to immobilize the leg. Local applications should be hot and not in the form of the ice bag, the traditional method.

High saphenous division cuts short the disease. Provided the thrombosis has not reached the saphenous opening, the usual high division can be performed. Following the operation, the patient may be allowed to get up early as after any high division, the leg being bandaged up to the knee.

Once thrombosis has occurred, whether or not it has occupied the whole length of the great saphenous vein, and has been treated in an ambulatory way or by rest in bed, there is a tendency to recurrence. High division, before another attack can occur, may properly be performed. Such treatment usually restores the individual to active service.

RUPTURE OF VARICOSE VEIN

This may occur unexpectedly at any time. The leak occurs below the knee at a point where thin skin covers the vein. Immediate treatment consists in elevation of the leg and local application of a pad and firm bandage from the toes to the region of the rupture. Later, high division of the varicose vein should be made.

THROMBOSIS

Thrombosis in the veins of the lower extremity is a common complication of injuries of many sorts, of operations, fractures and serious diseases—any conditions in fact which injure, weaken and impose life in bed. Very rarely, it occurs in young persons of either sex during active life, usually after some trifling strain or such an accident as fracture of a metatarsal bone. There are, broadly speaking, two forms which merge into one another: (1) an inflammatory, obstructive form, thrombophlebitis, the common “phlebitis” or “milk leg” of women, that is, phlegmasia alba dolens; and (2) a bland, nonobstructive form, the common cause of a soft thrombus and of pulmonary embolism, sometimes called “phlebothrombosis”

Thrombophlebitis

This obstructs the femoral and often the external iliac vein. It may commence in the upper part of the femoral vein—painful from the start and the cause of a swollen, white leg. Nevertheless it may originate in the deep veins of the lower leg, mounting, with little notice, to the upper portion of the femoral vein before the disease becomes outspoken. Usually, associated with femoro-iliac thrombophlebitis, there is a perivascular inflammatory reaction causing tenderness and induration over the femoral vessels at the groin. The perivascular inflammation also leads to arterial spasm, both locally, in the main artery, and reflexly in the periphery of the limb. There is thus in many cases a diminished arterial flow, which may amount to serious ischemia and even, very rarely, gangrene of the limb. On the other hand, if peripheral spasm predominates, there is a widespread edema and some degree of cyanosis. Edema is by far the more common. It is these reactions which have led to treatment intended to relax vascular spasm, that is, by sympathetic procaine block and heating the body.

Thrombophlebitis usually starts within a few days to two weeks after the injury or operation. Pain is referred to the groin, thigh and back of the knee, that is, along the femoral vessels. Swelling appears early and involves both leg and thigh. There are often several degrees of fever. Depending on the extent of the thrombosis and the amount of perivascular inflammation, the disease may run its course in two weeks or drag on for months with little danger of pulmonary embolism. It usually leaves behind a tendency to edema on long standing or exertion, and often unaccountable and obstinate indurations, local edemas, pigmentations and, in the end, ulcers of the lower leg. Thus it often renders the individual unfit for military service.

Treatment.—The limb should be elevated, by raising the foot of the bed, but is better not immobilized. Indeed, it should gently be exercised as much as its soreness and heaviness allow. The ice bag, save for relief of discomfort, is useless. Local heat to the groin and warming the body, to secure vasodilatation, are far more desirable.

Sympathetic procaine block (Figs. 12, 13) is very useful, especially in the cases in which there is much edema and pain. The treatment should be given early and may be repeated on several occasions. Swelling is sometimes dramatically relieved and subsequent complications are decidedly less frequent and serious.

Small doses of roentgen rays, not exceeding 100 r units, hasten the absorption of the periphlebitic exudate.

As fever, pain, and edema disappear, exercise out of bed is gradually begun and increased. Long hours of standing should be avoided, and elevation should be used to control any recurrent swelling.

Thrombosis without Obstruction (Bland Type), Phlebothrombosis

This disease is treacherous. It may show no noticeable signs until it causes fatal embolism. Almost invariably, it occupies some part of the many large venous plexuses among the muscles of the calf. From here it sometimes grows into the popliteal and up the femoral vein as a nonobstructing, floating thrombus, easily broken off. Often it remains entirely local. Or it extends in an adherent, but usually not fully obstructive, form well up into the femoral and even the iliac veins. From this region emboli of moderate size may repeatedly break off. Thus repeated pulmonary embolism is likely to come from the external iliac vein and a long, fatal embolus from the lower part of the femoral vein. That this locally quiet disease merges into the inflammatory obstructive type should be understood.

Clinical Features.—In active life, the earliest sign is usually slight lameness on walking. Tightening or stretching the great flat muscles of the calf is painful. The ankle is often a little swollen, the foot slightly cyanotic. All such signs quickly disappear when the individual goes to bed, but recur when he gets up. There may or may not be tenderness in the leg or foot. But there is always discomfort back of the calf or knee on forcibly dorsiflexing the foot—*dorsiflexion sign*.

When the disease appears in the course of life in bed, all such signs are less noticeable. Pain may or may not occur, swelling is absent and only a little blueness appears when the leg hangs from the edge of the bed. Local tenderness, and especially the dorsiflexion sign, are most likely to confirm the diagnosis.

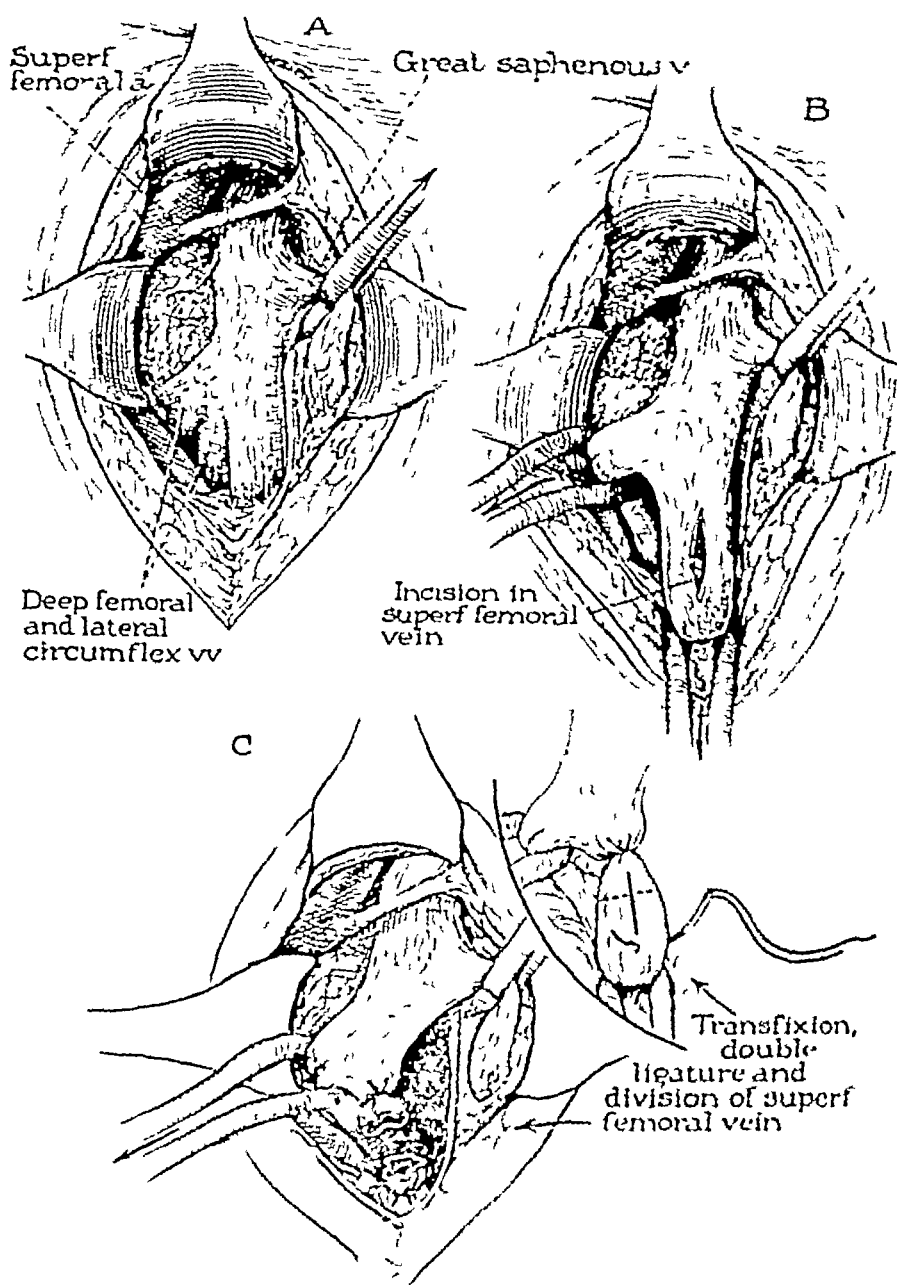


Fig. 20—Exploration and division of superficial femoral vein. A, The exposure. The great saphenous vein is the guide. B, All branches being controlled with rubber tubing, the superficial femoral vein is opened. No thrombus is found or, if present, is removed by suction. The vein is ready for division. C, The vein is doubly ligated, transfixed (inset), and divided just distal to the profunda femoris.

The course of the untreated disease in pulmonary embolism in unoperated cases, those in which the patients are m

and recovery, but it ends in death in many cases, particularly in those of advanced years of age.

Treatment.—For individuals less than fifty years of age, confinement to bed, elevated 6 inches (15 cm.) at the foot, but without immobilization of the leg, is usually curative. In ten days or so, the local signs will entirely have disappeared. Then, for several more days, the leg should be exercised freely in bed. If, when the individual begins to walk, in a semi-elastic bandage, no lameness reappears, swelling is slight, and cyanosis absent, the disease may be believed cured. But if the signs reappear, recurrence surely has taken place and division of the femoral vein usually should be undertaken.

Surgical treatment is intended to forestall or prevent the occurrence of embolism. Exploration of the femoral vein at the groin is illustrated in Fig. 20. The incision may be oblique, that is, parallel to the inguinal ligament for 5 to 6 inches (13 to 15 cm.) or longitudinal. The saphenous vein is the guide to the femoral. Under temporary control by rubber tubing, the superficial femoral vein is explored and, if free of thrombus at this level, is divided between double silk ligatures (one on each stump should transfix the vessel). If loose thrombus is found, it may be sucked out and division made as if the vein had been found empty.

If adherent thrombus is found, with thrombosis in the profunda femoris, the femoral vein may be divided. This causes serious venous congestion in the leg. Probably less congestion will result, and the procedure will more effectively prevent embolism, if the external or common iliac vein is approached and divided extraperitoneally through an incision as for ureteral exploration (collateral veins are most abundant and efficient in case of division of the common iliac).

Thrombosis in Superficial, Nonvaricose Veins

This is a freakish process which crops up unexpectedly as a result of moderate injuries. It tends to remain local and rarely leads to embolism. The thrombosed vein is usually sore and slightly inflamed. Thrombosis may heal in the course of a few weeks and jump to a new spot, usually proximal to the first (wandering phlebitis phlebitis migrans). In that case it may be evidence of thrombo-angiitis obliterans (which see, p. 243).

Treatment calls for elevation in bed, as for thrombosis in a varicose vein. Cure is usually rapid, but proximal (high) division of the vessel probably gives the promptest relief and permits early resumption of an active life.

Thrombosis of Axillary Vein, Thrombosis by Effort

The cause of this unusual form, which occurs in vigorous, active individuals at any age, is the strained use of the arm in an elevated

position; rarely cranking an automobile, or a fall. It is believed that if the breath is held (filling the subclavian and axillary veins), use of the arm, as it is held overhead, injures a large valve in the axillary vein or a portion of the wall of the vein. This is a pure thrombosis, limited in extent and with little tendency to embolism.

The clinical signs of axillary thrombosis are cyanosis and swelling of the corresponding arm. The veins of the shoulder are especially prominent. Pain is not a feature. The obstruction persists for several weeks as a rule. Then gradually recovery follows, leaving little or no disability. Only very rarely, some pain and edema, as after a femoro-iliac thrombophlebitis, persist.

Treatment—Elevation of the arm on a pillow, as the individual lies in bed, is usually sufficient. Use of the arm is resumed as the signs disappear. In the rare event of persistent pain and edema, sympathetic procaine block at the stellate ganglion gives relief. The thrombosed segment of the axillary vein may also be resected. The prognosis for a useful arm and active service is good.

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